The American Journal of Surgery

EDITED BY THURSTON SCOTT WELTON, M.D.

April 1942

Symposium

DIAGNOSIS IN SURGICAL CONDITIONS

Edited by

JAMES T. PRIESTLEY, M. D.

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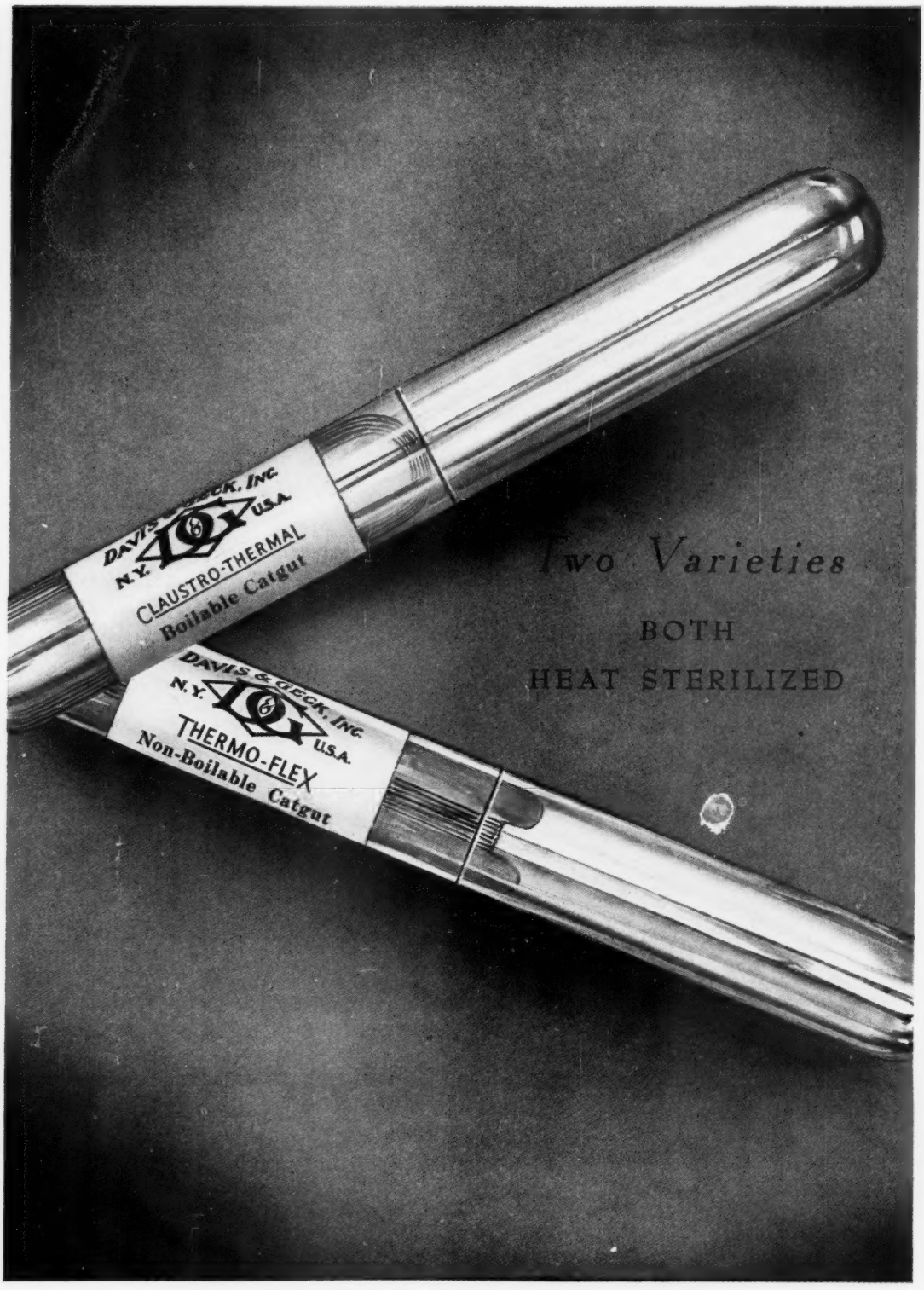

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A ligature can fail to fulfill its function for two reasons—the ligature slips before the fibrous tissue forms; or it causes necrosis and cuts through the wall of the vessel.

Any unanchored ligature, regardless of size or type of material, can slip off the end of a blood vessel unless it is tied so tightly that constriction occurs. If marked constriction occurs, necrosis takes place and the ligature may cut through producing secondary hemorrhage or an hematoma. Only transfixion of the ligature or anchoring of it to the surrounding tissue definitely assures that the ligature will remain exactly at the point where it was tied, and with transfixion the necessity for tight tying vanishes.

Whether to employ transfixion is sometimes determined by the size of the vessel to be ligated. For example, transfixion is not required if it is believed that the fibrinous clot will produce hemostasis.

The material employed for the ligature should have enough strength to satisfy the mechanics of setting a reasonably firm knot. The mechanics vary with the location of the vessel. Less traction is required to set a knot on the surface than in the depths of a cavity. Under the latter circumstance, a ligature of larger size must be used.

If a ligature is tied tight enough to cause necrosis, bacterial infection is apt to occur in the area of constriction. Ligating large tufts of tissue after crushing the bleeding points with blunt pointed hemostats also provides suitable soil for bacterial growth. Fine pointed hemostats should always be used and only the exact bleeding points clamped and ligated. When bacterial infection occurs in a wound for any reason a blood vessel may be eroded.

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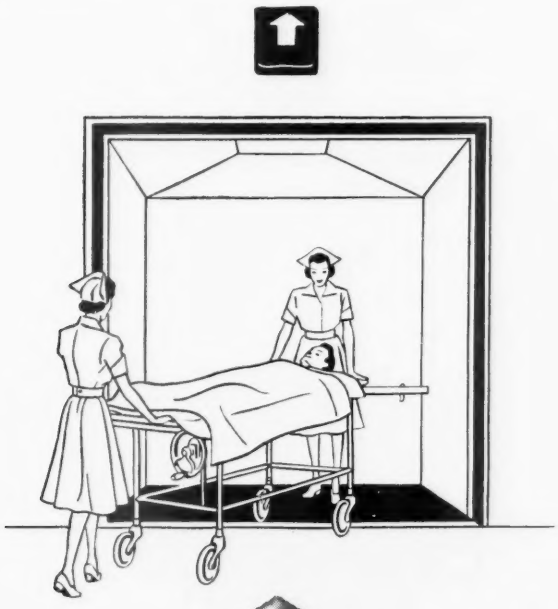

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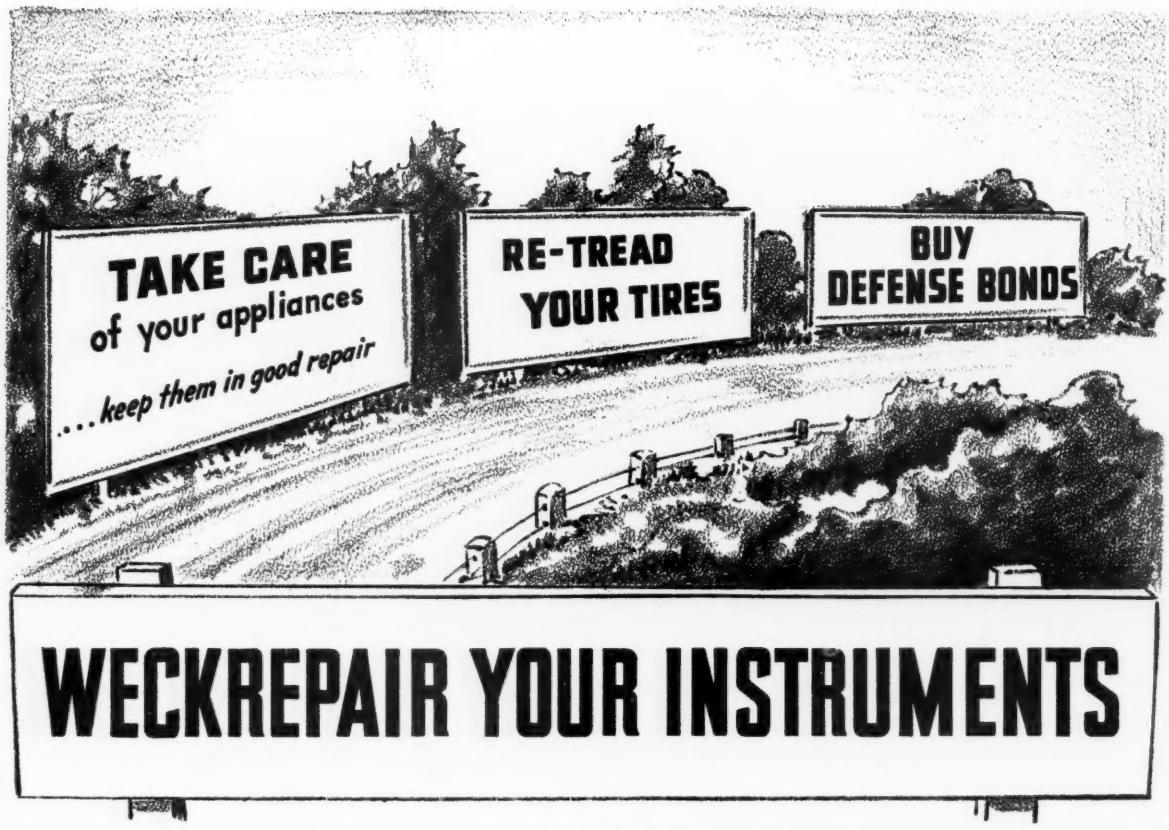

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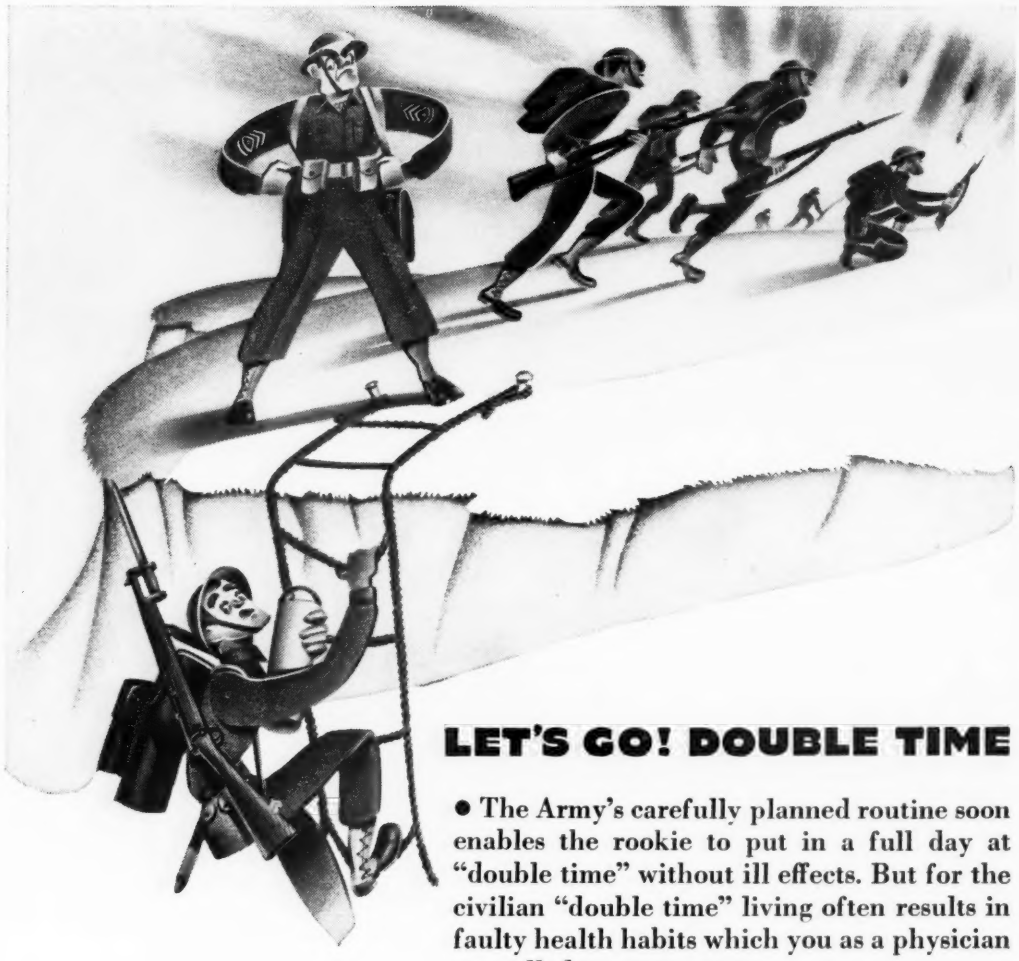

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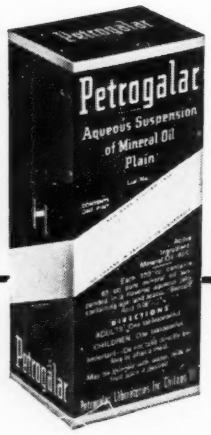

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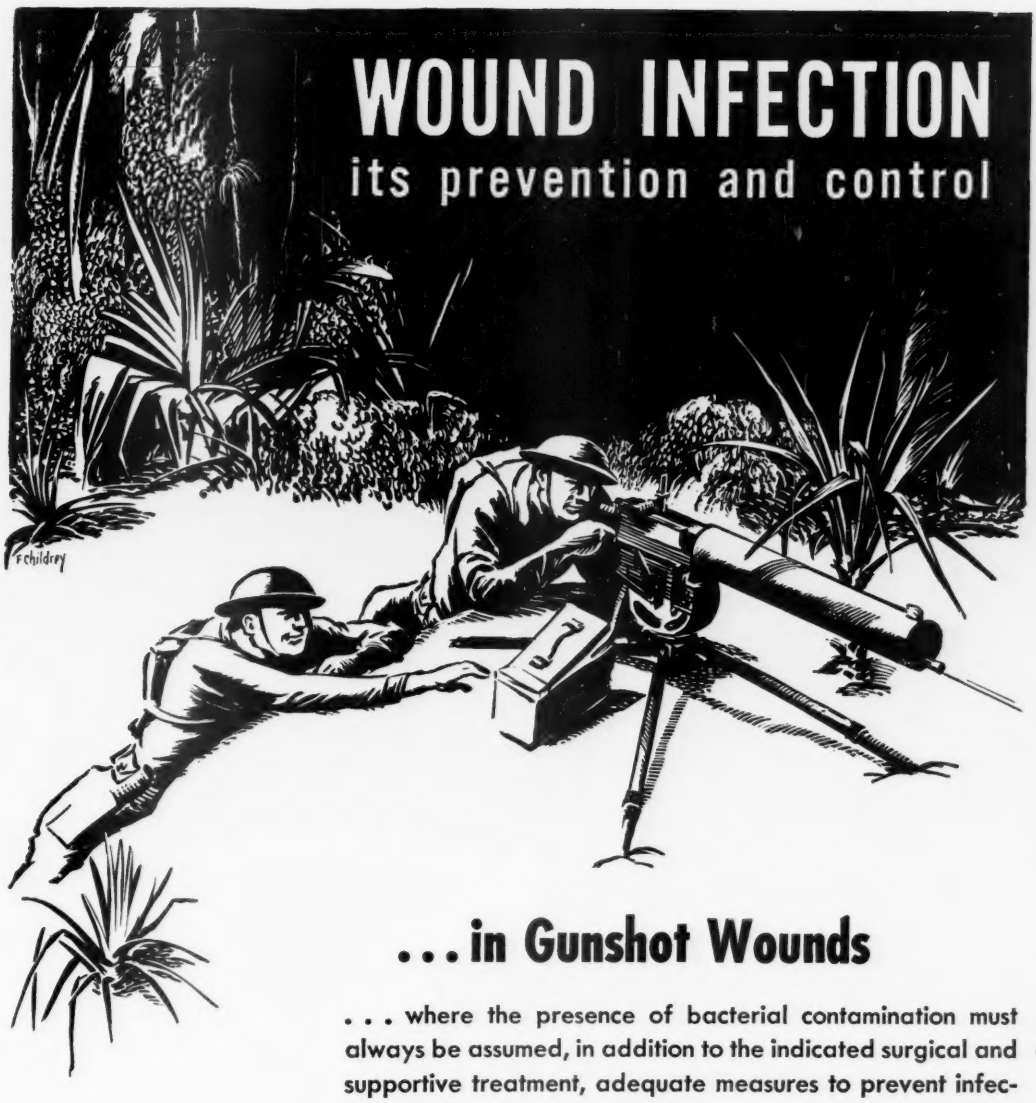

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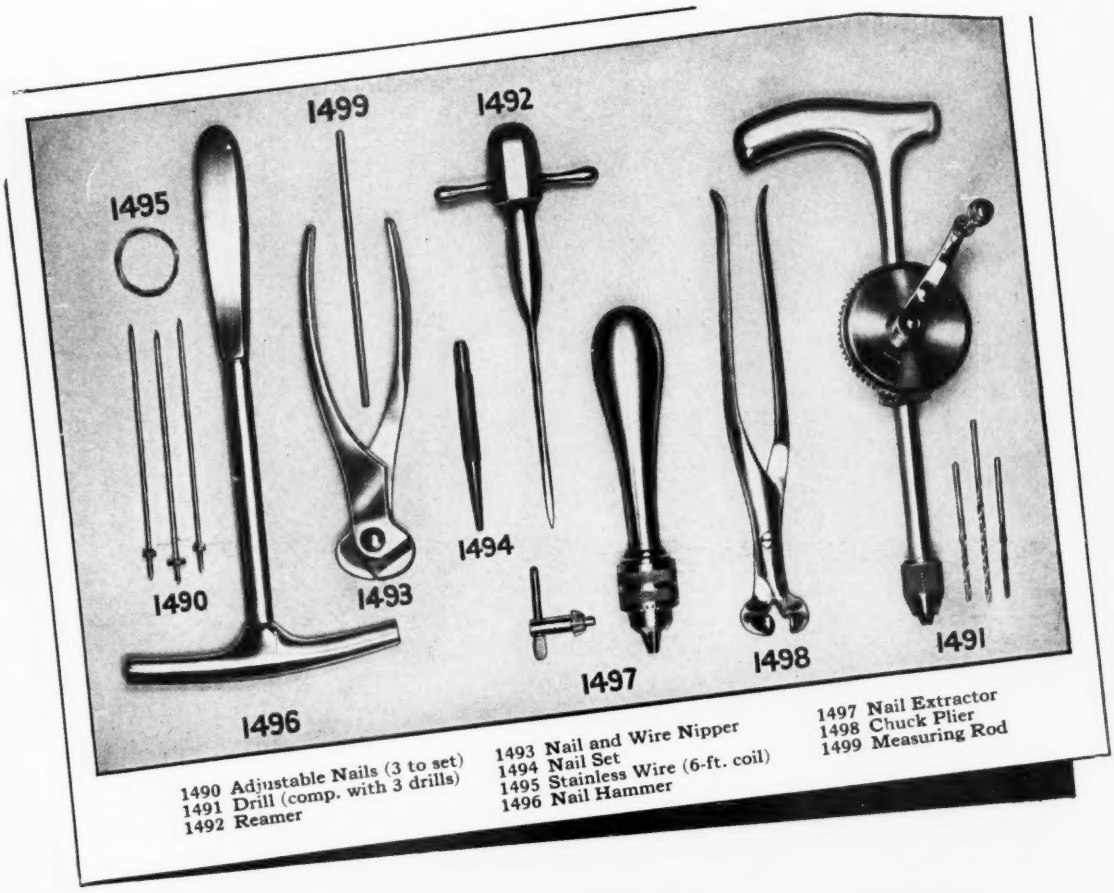

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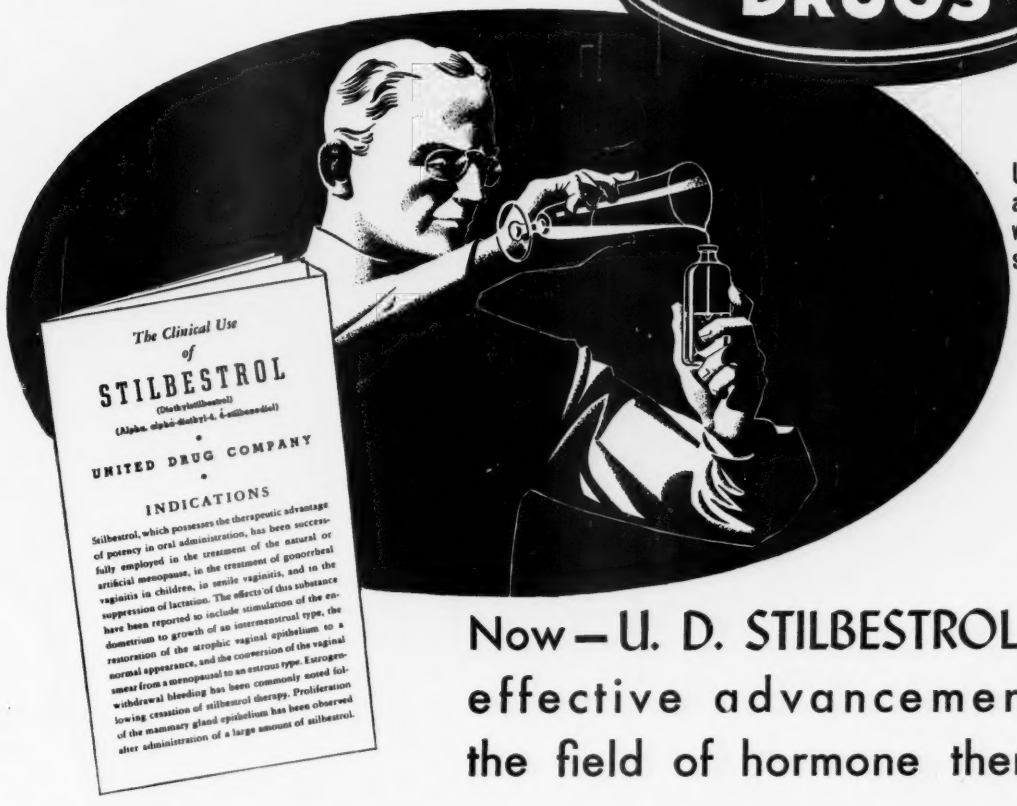
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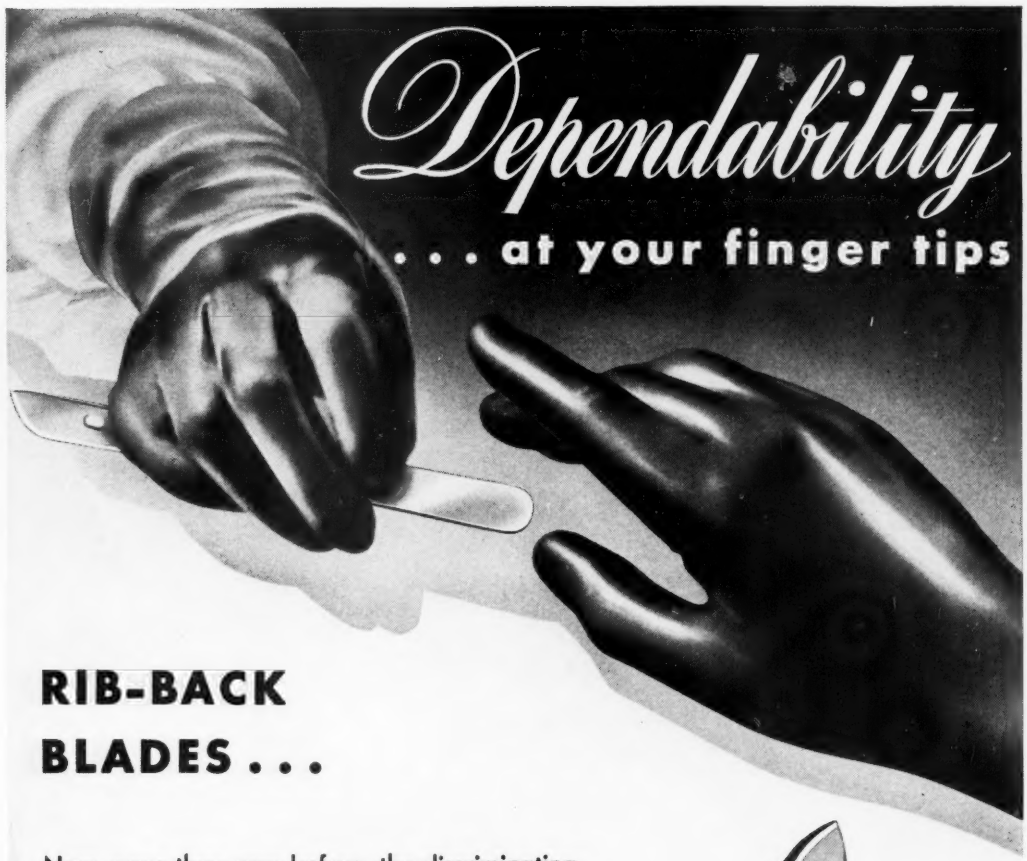

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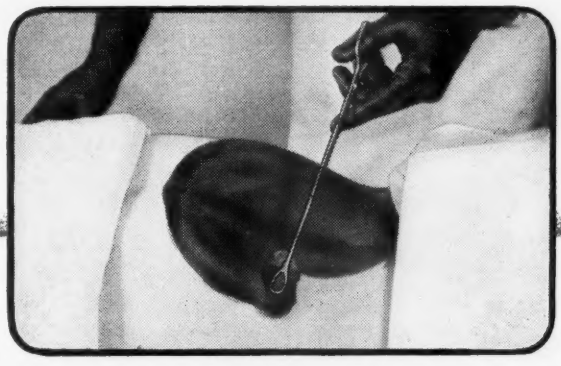

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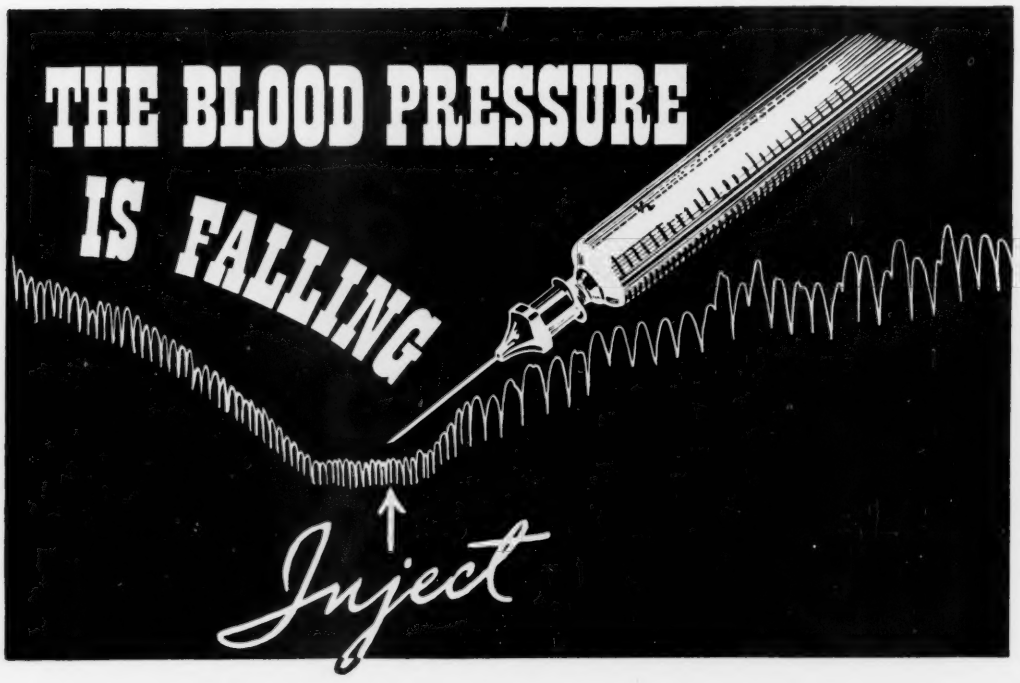

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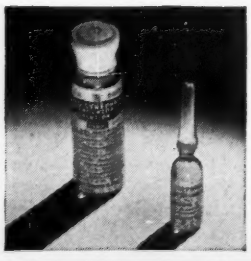

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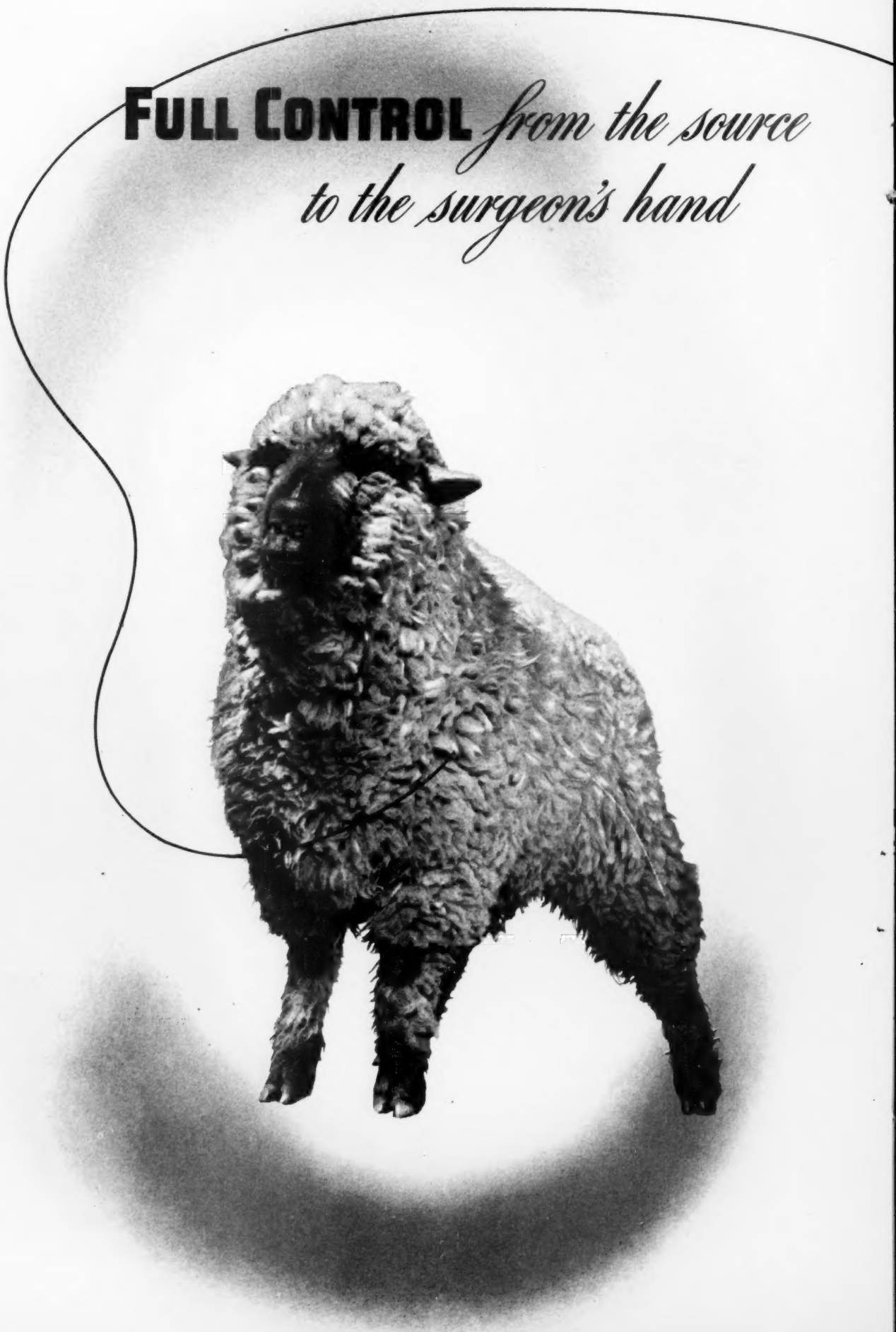

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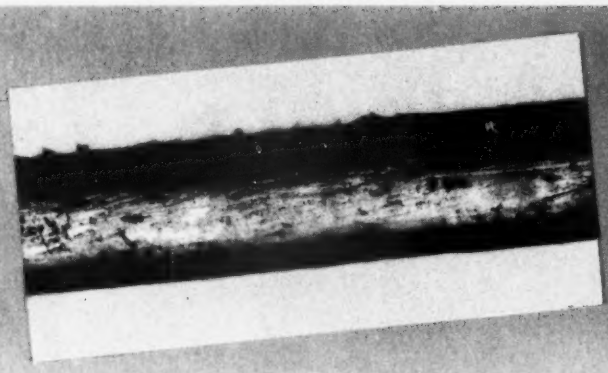

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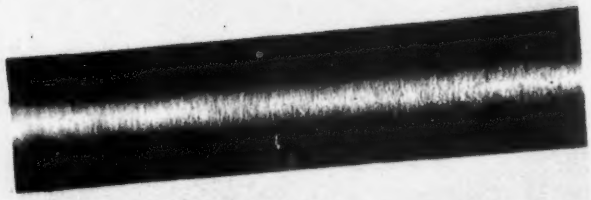

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A PRACTICAL JOURNAL BUILT ON MERIT

NEW SERIES VOL. LVI APRIL, 1942 NUMBER ONE

Editorial

THOROUGHNESS IN DIAGNOSIS

CALEB HILLIER PARRY, first to describe hyperthyroidism, spoke to his students as follows: "There is one malady which I have in five cases seen coincident with what appeared to be enlargement of the heart, and which, so far as I know, has not been noticed, in that connection, by medical writers. This malady to which I allude is enlargement of the thyroid gland.

"The first case of this coincidence which I witnessed was that of Grace B., a married woman, aged thirty-seven, in the month of August, 1786. Six years before this period she caught cold in lying-in and for a month suffered under a very acute rheumatic fever; subsequently to which, she became subject to more or less palpitation of the heart, very much augmented by bodily exercise, and gradually increasing in force and frequency until my attendance, when it was so vehement that each systole of the heart shook the whole thorax. Her pulse was 156 in a minute, very full and hard, alike in both wrists, irregular as to strength, and intermitting at least once in six beats. She had no cough, tendency to fainting, or blueness of the skin, but had twice or thrice been seized in the night with a sense of constriction and difficulty of breathing, which was attended with the spitting of a small quantity of blood. She described herself also as having frequent and violent stitches of pain about the lower part of the sternum.

"About three months after lying-in, while she was suckling her child, a lump about the size of a walnut was perceived on the right side of her neck. This continued to enlarge from the period of my attendance, when it occupied both sides of her neck, so as to have reached an enormous size, projecting forward before the margin of the lower jaw. The part swelled was the thyroid gland. The carotid arteries on each side were greatly distended; the eyes were protruded from their sockets, and the countenance exhibited an appearance of agitation and distress, especially on any muscular exertion, which I have rarely seen equalled. She suffered no pain in her head but was frequently affected with giddiness.

"For three weeks she had experienced a considerable degree of loss of appetite and thirst, and for a week an edematous swelling of her legs and thighs, attended with very deficient urine, which was high-coloured and deposited a sediment. Until the commencement of the anasarcous swelling she had long suffered night sweats which totally disappeared as the swelling occurred. She was frequently sick in the

morning and often threw up fluid tinged with bile."

At the present time when laboratory procedures of all types figure so largely in clinical diagnosis, it seems appropriate to recall the limited facilities for investigation which were available to physicians of earlier generations. Despite this fact, most of the clinical entities known today were recognized by these men through careful consideration of the history and keen utilization of the senses of sight, hearing and touch. The inestimable value of laboratory investigation in the recognition of disease is not to be denied, but dwarfing of acumen which may result from too great reliance on these data should be avoided.

The absolute necessity of accurate diagnosis as a prerequisite to intelligent treatment is recognized by all engaged in the practice of medicine. If one quality of the examining physician was to be emphasized, which probably is more responsible for accurate recognition of disease than any other single factor which he might possess, it would be *thoroughness*. Without thoroughness in his examination even the most astute clinician or surgeon will fail in certain cases to detect perhaps the one symptom or clinical finding which might lead to the true diagnosis. Thoroughness requires time which even the busiest and most hurried physician must be willing to spend if error is to be avoided. Accurate diagnosis includes not only the recognition of full-blown disease, when it is presented as such, but also the detection of any pathologic process in its earliest stage when treatment can be administered most effectively. Unfortunately, the descriptions of many diseases in numerous textbooks emphasize too greatly abnormalities which are present only late in the progress of the disease. To offer the best opportunity for early and permanent cure disease must be recognized at its onset, in its early stages, before complications and distant effects make its presence obvious to the most inexperienced. Little good is afforded the patient by recognition of a primary malignant growth through secondary metastatic developments. Great emphasis should be accorded early symptoms in teaching the undergraduate medical student. He will become all too familiar with the findings of far advanced disease following his graduation.

On behalf of the Editorial Board of the American Journal of Surgery I wish to express our appreciation to all the authors who contributed the excellent and instructive articles presented in this issue of the Journal.

James T. Priestley, M.D.

War Surgery and Traumatic Lesions

EARLY DIAGNOSIS OF CRANIOCEREBRAL INJURIES*

DONALD MUNRO, M.D.

Surgeon-in-Chief, Boston City Hospital; Assistant Professor of Neurological Surgery, Harvard Medical School and Associate Professor of Neurological Surgery, Boston University Medical School

BOSTON, MASSACHUSETTS

IT is, or should be, axiomatic that diagnosis is no more than a word picture of the pathological condition produced by disease or injury. In injuries this condition is organic. Moreover, it is indisputable that objective signs will be of greater diagnostic import than subjective symptoms and that the less such signs are subject to individual variations of interpretation by the physician and patient the greater is their diagnostic accuracy. Such sweeping generalities must be qualified by the proviso that the indicated pathological changes are known and not merely inferred and that it is possible to demonstrate reliable objective variations from the normal. In this particular problem, it is also true that when attempts are made to use subjective symptoms or objective signs whose significance depends on alternative interpretations by the physician, great care must be exerted to avoid ascribing diagnostic significance to what are actually lateralizing or prognostic data.

An appreciation of the above diagnostic fundamentals is of more than usual importance in dealing with head injuries. Physicians and surgeons who have been trained in the more leisurely schools of neurology and tumor neurosurgery are frequently at a loss in the hurly-burly of craniocerebral injuries in which decisions have to be made rapidly and without benefit of the detailed meticulous study that tradition has decreed must precede the diagnosis of intracranial disease. Moreover, the clouding of the picture by associated injuries, rapid intracerebral changes that may not only vitiate otherwise reliable signs but actually apparently reverse their significance, and complications such as surgical shock and dehydration lead to profound diagnostic errors, ill-conceived therapy and higher mortality. There is a great need for sanity and objectivity in the diagnosing of the intracranial pathological conditions that result from acute craniocerebral injury.

General Considerations. Before anything further can be done in the diagnosis of a craniocerebral injury, the surgeon must first satisfy himself that not only has the patient sustained a blow on his head but also that the blow has been of sufficient magnitude to produce intracranial pathological conditions. I do not mean to underestimate the importance or significance of lacerated wounds, contusions or hematomas of the scalp. They are neglected here only because of lack of space. On the other hand, I am sure that a fracture of the fault or base of the cranium can never occur in the absence of some degree of brain injury if the latter is properly and promptly looked for.

The fact of the patient's having sustained a blow on the head will be demonstrable by the presence of a bruise, cut or hematoma of the scalp, by the observation of reliable witnesses or by the demonstration of a fracture of the skull. Except in what may be classed as perforated wounds of the skull, the significance of the blow in terms of brain injury will depend upon the demonstration of loss of consciousness or a

* From the Department of Neurosurgery of the Boston City Hospital.

period of amnesia covering all or part of the time of the accident and extending a variable distance antecedent to and after the injury. Such amnesia results from and predicates unconsciousness.

Consciousness is a relative term that has to do with one's awareness of his surroundings. No person can be completely conscious at any given instant. Loss of consciousness predicates a decrease in one's awareness of one's surroundings. At one extreme the patient is completely out of touch. On recovery of consciousness he will be forever unable to fill in this void in his existence and will only then recognize it as having been a void. On the other extreme, the patient will be so little out of touch that until checked by the observations of impartial witnesses he will be unaware that any void has existed. Neither condition requires immobilization or necessarily unusual or abnormal activity on the part of the patient, although neither obviates such conditions. Demonstration of the first will be all too apparent when the patient is asked to do something unusual while unconscious, or on recovery of consciousness, has an apparent amnesia. The classical and common example of this is found in the football player who, after being kicked on the head at the start of the fourth quarter and after being removed ten minutes later because of a failure to hold onto a forward pass that was thrown into his arms, was unable to find his way to the locker room and still later inquired as to what team he had been playing against and what the score was at the end of the third quarter. Demonstration of the second may be extremely difficult and may involve the interviewing of others beside the patient. From these witnesses data must be obtained relative to associated facts and incidents that might have or did happen at the time of the accident. Later cross-questioning of the patient in regard to these specific proved instances must then be carried out in an endeavor to demonstrate decrease in his awareness of these surroundings. Many such patients will admit to having been "dazed," "woozy," "groggy," "dizzy," or other similar conditions while at the same time they will vehemently deny having been "unconscious." It should be noted that regardless of the degree of unconsciousness, the time factor is of little diagnostic significance. I am convinced that unconsciousness as such has no more diagnostic significance than that its presence demonstrates that a blow severe enough to produce some degree of brain injury has struck the victim's head. On the other hand, its true absence—not an absence that depends for its proof on such data as the unsupported opinion of the victim or his failure to develop physical immobility—indicates an absence of brain injury except in perforated wounds of the skull and brain. This, as has been pointed out by Denny-Brown and Russell,[1] is dependent upon the relative mass of the striking object and the head, as well as the velocity of the striking object. The smaller the mass, the more local is the effect and hence the less the likelihood of the production of unconsciousness. Changes in consciousness occurring later and in the course of treatment are prognostic and not diagnostic. For practical purposes, I believe that one may consider a patient who has either an obvious scalp injury, fracture of the vault or base of the skull or a reliable history of a blow on the head as having been struck on the head. If, in addition, one can demonstrate any degree of loss of consciousness, regardless of its length or severity, and especially if it is or has been associated with amnesia, the surgeon must conclude that the blow has been severe enough to produce some degree of brain or intracranial pathological condition.

NONOPERABLE GROUP OF ACUTE CRANIOCEREBRAL INJURIES

Those acute cerebral injuries the pathology of which is such as not to call for operative therapy are concussion, edema and congestion, contusion and/or laceration and perforating wounds of the skull. The analogous cranial conditions are those

in which the condition consists of a linear or diastasic or comminuted fracture of the skull that is *not* compounded and that neither enters an air sinus nor the nose and that has not torn into any of the large venous sinuses, the middle meningeal or the internal carotid artery. These latter will always be accompanied by some degree of brain injury and the diagnosis should always include a description of this important part of the craniocerebral pathological condition.

Acute Nonoperable Cranial Injuries. The diagnosis of the acute nonoperable cranial injuries is not difficult. Simple linear or diastasic or comminuted fractures of the vault are only diagnosable by *x*-ray examination. To be accurate and significant the films must include right and left lateral stereoscopic views. Single films are not conclusive except in certain special instances. Since the demonstration of such fractures has no therapeutic significance, *x*-rays taken for the purpose should always be postponed until after the patient has recovered consciousness and is again co-operative. Fractures into the nose by way of the cribriform plate are usually not diagnosable by *x*-ray. Their presence is recognized by the leakage of cerebrospinal fluid from the nose or into the posterior nasopharynx. Such a diagnosis calls for therapy at the earliest possible moment if the patient is to be saved from meningitis. The presence of a fracture into any of the air sinuses should be suspected whenever there is prolonged bleeding from the nose just as fractures into the ear should be suspected when there is excessive bleeding from the latter. Of course, local damage to both nose and ear must first be eliminated as the cause of the hemorrhage. Battle's sign—a subcutaneous hematoma that develops in the region of the mastoid an appreciable time after the receipt of the injury—is probably pathognomonic of a fracture extending into the mastoid. Adequate *x*-ray studies will demonstrate most of the fractures into the air sinuses and many but not all of the fractures into the petrous ridge. The presence of a suspected fracture of the posterior wall of the frontal sinus must be verified as soon as possible because if there is reason to suspect that the mucous membrane of the sinus has been torn in addition to or because of the fracture, the latter must be considered to have been compounded into the nose with all that that implies in the line of treatment. This is one of two conditions in which diagnostic *x*-ray studies are imperative at the earliest possible moment. The other is whenever the surgeon has reason to suspect the presence of an extradural hemorrhage. In both instances, because the diagnosis may have to be made before the patient is co-operative the surgeon will usually have to be content with a single film. To have this film satisfactory and of diagnostic significance, he must be prepared to tell the roentgenologist the area of the skull that he wants to study, must be willing to assume the responsibility for moving the patient to an *x*-ray room and must co-operate with the roentgenologist in holding recalcitrant patient's heads quiet by force if necessary during the exposure. In return for that the surgeon can expect from the roentgenologist a film that will show not only the fracture line but also the shadows of the grooves of the middle meningeal artery, the superior sagittal and the lateral sinuses, and the posterior wall of the suspected frontal sinus. Fractures that involve the cavernous sinus and the internal carotid artery are diagnosable only by inference, being usually invisible by *x*-ray. Their presence can be inferred whenever a unilateral orbital bruit or pulsating exophthalmos develops a short time after a head injury. In the absence of such an arteriovenous fistula, the fracture is undiagnosable except by chance.

Acute Nonoperable Cerebral Injuries. In discussing the diagnosis of acute nonoperable cerebral injuries, it is worth while to consider in some detail the pathological condition spoken of clinically as concussion. This is the least severe result of a blow on the head. It is always present, however,

in association with more severe and more lethal generalized traumatic cerebral conditions. The pathology of concussion is not known in all details. Denny-Brown and Russell[1] have presented experimental evidence to show that in animals the phenomenon of concussion which they define as "direct traumatic paralysis of nervous function without vascular paralysis" can only be produced "by subjecting an animal's head to a sufficiently high rate of change of velocity or by a crushing injury such as striking the head while it is supported on a hard surface." The first they call "acceleration concussion" and the second "compression concussion." They claim that the paralysis of concussion is immediate and antedates any vascular changes that take place even as the result of vascular stress, except for "an increased blood flow through the brain and brain stem." In addition, they found that the vagoglossopharyngeal system is often stimulated by subthreshold blows with resultant depression of cardiac, vasomotor and respiratory function for short periods. Also that when failure of blood pressure results from severe head injury, the mechanism appears to be identical with that of primary surgical shock—"and is associated with peripheral vaso-constriction and paralysis of the venous side of the circulation." Clinically, my experience leads me to believe[2] that in man alteration in the cerebral arteriolar and capillary circulation is perhaps coincidental with the later phases of such concussion and, in any event, may follow after so closely as to be undistinguishable as part of the phenomenon. To be sure, these conclusions are inferential rather than evidential but even so have the merit of having served as a satisfactory basis for the therapy applied to the craniocerebral injuries seen during the past ten or more years. These latter changes are apparently initiated by the vasovagal reflex and, in any event, are associated with unconsciousness.

With their return to normal, consciousness also returns and there is no longer either subjective or objective evidence of any cerebral pathological condition, that is, the patients have neither signs nor symptoms of an injury. Because, as far as is known, no patient has ever died while suffering from clinical concussion alone, the condition cannot be more certainly identified. Also, inasmuch as the period of unconsciousness is always short, often being a matter of seconds, direct observations in men have not been made during that time in regard to intracranial pressure, the chemistry of the cerebrospinal fluid, and so forth. Furthermore, other than the associated unconsciousness, the vascular changes if present produce no signs or symptoms. The diagnosis, therefore, depends first upon the demonstration of a blow on the patient's head which produced unconsciousness and second upon the further demonstration that on recovering consciousness the patient complained of no symptoms and on extended examination had no signs of any injury, with the occasional exception of damage to the scalp or fracture of the skull. In other words, as far as the patient knows on recovery of consciousness, he is well and normal. The diagnosis of clinical concussion uncomplicated by anything except a scalp injury or simple fracture of the skull can be made only under such conditions. If and when made, it is obvious that being well, the patient requires no treatment, no hospitalization and no convalescent care and cannot have, by any stretch of the imagination, a "postconcussion state" that has any connection whatsoever with his head injury.

When the injury to the patient's head is more severe, however, further pathological changes take place in the brain and further diagnoses must be added to that of concussion. These are, in order of severity, edema and congestion, contusion and laceration of the brain. Each succeeding one is added to all the preceding less severe conditions. All varieties of subjective symptoms occur in any combination in all of these conditions. In general, however, it may be said

that to the unconsciousness of concussion is added the headache and dizziness of edema, the disorientation, confusion or stupor of contusion and the coma of laceration. Because the brain is affected as a whole even although the maximum damage may be limited to one area all conceivable combinations of reflex, sensory and cranial nerve abnormalities may be found in any given case. Such wide variations as hemiplegia and Jacksonian convulsions in children with cerebral edema, and supernormal activity without somatic reflex changes in adults with lacerated brains are frequently seen. I have even seen such physical changes as are usually considered to indicate decerebration clear up after nonoperative therapy and the patient leave the hospital in essentially normal condition. To be sure, such classical signs as the Babinski, increased reflexes in a paralyzed limb or combined facial and acoustic nerve paralysis occurring on the same side have certain localizing values but cannot be considered diagnostic of the pathological cerebral condition that underlies them. Edema of the optic nerve heads points only to increased intracranial pressure. Fixed dilatation of one pupil occurs not only in nonoperable but more usually in operable conditions and although considered by some to be of lateralizing significance has not proved to be so in my experience. Bilateral fixed dilated pupils is a prognostic rather than diagnostic sign and is commonly seen just before death. Increasing coma or a trend toward coma from confusion or disorientation is only prognostic and indicates unfavorable progress and the need for more and increasingly accurate diagnostic studies.

How then can one diagnose these conditions? The answer to this question is found in a knowledge of the changes in normal cerebral physiology that the pathological condition there present has produced. Just as unconsciousness following a head injury is the diagnostic touchstone for concussion, so too edema and congestion, and contusion and/or laceration have their own peculiar and recognizable additional objective signs. When edema and congestion are present there is, among other things, an increase in the brain volume, a rise in intracranial venous pressure and often petechial and coalescing intracortical hemorrhages. The cerebrospinal fluid pressure is forced up until such time as the intracortical condition returns to normal, but the cerebrospinal fluid itself remains unchanged both as to cell content and chemistry. Thus, the condition can be diagnosed by demonstrating an increased intracranial pressure and normal cerebrospinal fluid in a patient who has been knocked unconscious by a blow on the head. This is easily done by a lumbar puncture during which the pressure is measured by an appropriate manometer and sufficient cerebrospinal fluid collected for chemical and cytological studies. Other things being equal, this is the only way, in my experience, in which this diagnosis can be made from indisputable facts. Of course, the findings will be normal if this examination is carried out after the edema and congestion have subsided.

Contusion and/or laceration, as pointed out above, are associated with and are present in addition to clinical concussion and edema and congestion. In my experience they cannot and do not occur separately, although Denny-Brown[3] and Eden and Turner[4] claim that contusion does do so. I believe that apparent absence of concussion and edema can be accounted for, except in perforating wounds, by the willingness of the surgeon to accept the patient's unverified word as to unconsciousness and to the absence of factual data as to the presence of complicating dehydration. The diagnostic criteria then must include unconsciousness following a blow on the head and the demonstration of an increased intracranial pressure. The cerebrospinal fluid, however, will differ and will no longer be normal as to either cytology or chemistry. By definition, both contusion and laceration imply a rupture of surface vessels with escape of red blood cells into the cerebral subarachnoid spaces.

Since absorption of cerebrospinal fluid takes place from these spaces into the venous sinuses by way of microscopic openings in the walls of the arachnoidal villi or Pacchionian bodies; and since these openings are too small to allow the passage of red-blood cells, they shortly become plugged to a varying degree by the free cells that have been loosed in the cerebrospinal fluid as the result of the contusion or laceration. This causes a backing up of the cerebrospinal fluid in the ventricular and subarachnoid reservoirs with a consequent further rise in intracranial pressure. In addition, examination of the cerebrospinal fluid will demonstrate an abnormal cell count, a change in color and an increase in total protein content. These changes will vary with the amount of free bleeding that has taken place into the cerebrospinal fluid. The color will range from the light pink fluid of the acute mild contusion to fluid that resembles pure blood and that is found in the fresh severe laceration. The decision as to whether a borderline case is contusion rather than laceration will vary with the individual surgeon's interpretation. Thus, contusion and/or laceration of the brain can be diagnosed and, in my opinion, only diagnosed, other things being equal, by demonstrating that the patient has been knocked unconscious by a blow on the head, has a higher than normal intracranial pressure and has a bloody cerebrospinal fluid.

Perforating Wounds of the Skull. Perforating wounds of the skull either with or without injury to the meninges and brain beneath the perforation are extremely rare in civilian practice. At the last analysis, they are compound fractures but because of their size, their frequent multiplicity and the nature of the perforating agent they can, in most instances, be considered to be uncontaminated and therefore in no need of débridement. Attention has recently been called to their importance in war by Eden and Turner[4] who speak of the cerebral injury as "focal contusion." They point out that such a contusion is not preceded by or even necessarily accompanied by any loss of consciousness. They and Denny-Brown and Russell[1] state that the explanation of the phenomenon is found in the fact that the brain as a whole is not affected by such wounds because the missile is small and because its velocity is such that the energy inherent in it is largely dispersed in its passage through the skull and yet is not enough to fragment the adjacent bone or force particles of scalp or head covering into the cranial cavity. As the size and velocity or energy of the missile increases the depth of penetration as well as the extent of the damage and hence the liability to symptoms attributable to involvement of the entire brain, increases also. Glass and small metallic fragments are said to be the common causes of such war wounds. Denny-Brown[3] and Denny-Brown and Russell[1] describe another type of local contusion without associated concussion as being caused by the "piston effect" produced by slightly larger missiles. These cause a sudden sharp rise and fall of intracranial pressure and more extensive but still localized cerebral damage. These diagnoses are made from the history, the demonstration of evidence of local cerebral contusion or laceration without associated generalized brain damage and the visualization of the metallic fragments by *x*-ray.

My experience has been limited to four instances of perforating wounds of the skull. One was caused by fragments of glass from an automobile windshield. Their presence was unrecognized for nine months after the accident and then only because they were found in the course of draining what until then had been thought to be a cortical abscess of unknown etiology. Two were stab wounds, in one of which a kitchen knife entered the cranial cavity by way of one orbit. In the other a pocket-knife blade had been driven through a child's parietal bone. In the last case another child had fallen head first onto the sharp end of a long upholstery nail. The nail had penetrated the skull and underlying meninges and

brain. Unconsciousness was present in the first case only. The diagnosis in the last three cases was not difficult because all the patients arrived in the hospital with the perforating implement still in place in the various wounds.

Bullet wounds must be considered as compound fractures and not as perforating wounds because, with only rare exceptions, their effect on the brain is widespread and in no way analogous to a "local contusion."

OPERABLE GROUP OF ACUTE CRANIOCEREBRAL INJURIES

The operable group of usual acute craniocerebral injuries includes depressed fractures of the skull, compound fractures (including bullet wounds) of the skull, extradural and subdural hematomas. It should be clearly understood and cannot be repeated too often that there is *always* some form of nonoperable acute cerebral pathological condition accompanying all of these operable injuries. Rarely it may be so fleeting as to be undiagnosable by the time the patient reaches the surgeon but no dependence can be placed on this fortunate occurrence and these diagnoses, when correctly made, must include a word picture of the associated nonoperable brain injury. This portion of the diagnosis is made in the same way and based on the same criteria that are described above under nonoperable cerebral injuries.

Acute Operable Cranial Injuries. Insofar as the skull injury is concerned, the diagnosis of the acute operable cranial injuries presents no great difficulty provided that the need for diagnosing the associated cerebral injury is kept always in mind. The simplest form is the depressed fracture. This diagnosis can be made only by *x*-ray examination and then only with the aid of stereoscopic lateral views and possibly some especially angled views. Attempts to make this diagnosis without *x*-ray films and by palpation have a 50 per cent chance of error because of the palpable similarity between these fractures and subperiosteal hematomas.

Compound fractures of the skull, except as mentioned above, occur only in the presence of a wound of the scalp and only then if all the layers of soft tissue, including the pericranium above the fracture line, are torn and the fracture brought into actual or potential contact with the outside world. An accurate diagnosis can be made only by palpation with the finger, after it has been rendered sterile, of the fracture line through the scalp wound. If the scalp wound is too small to permit this, it should be deliberately enlarged. If it has been sutured and the surgeon is unwilling to allow the responsibility for the diagnosis to rest on the man who sewed it up, the sutures should be removed and the wound examined after that. There is no substitute for the surgeon's sterile finger in making this diagnosis. Neither probes, lights, *x*-ray films nor any other method compares with the finger in efficiency or accuracy. The best way to sterilize one's finger is to put a dry sterile glove on over the unwashed hand taking care, however, not to contaminate its fingers or palm in doing so. Bullet wounds should be *x*-rayed not so much for the purpose of diagnosis as to locate the foreign body prior to attempts at treatment. These two operable cranial injuries always have associated with them some type of nonoperable cerebral injury. They may also occur either alone or together or in combination with extra- or subdural hematomas or both.

Acute Operable Cerebral Injuries. The most difficult condition to diagnose of all those connected with acute craniocerebral injuries is the extradural hemorrhage. Maltby and I[5] have recently considered this and other aspects of the problem in detail and I will deal only with certain of the more important features here. At best, the diagnosis can only be tentative until confirmed by exploratory operation or autopsy. In spite of a small percentage of error, the surgeon is obligated to make this diagnosis and institute immediate operative therapy whenever he elicits a pathognomonic history. This will be whenever

he can get a history of unconsciousness following a blow on the head followed by a period of consciousness, which in its turn is followed by another and slowly developing second period of unconsciousness. The diagnosis can be strengthened by an *x*-ray examination as described above, and by demonstration of a high intracranial pressure with normal cerebrospinal fluid. The clot can be lateralized by the demonstration of contralateral hyperreflexia with or without hemiplegia, Jacksonian convulsions, aphasia, asterognosis, hemianesthesia and the like. However, of all of these signs the most informative is the *x*-ray film. Large clots that develop slowly will produce ipsilateral hemiplegia and reflex changes; clots that are formed as the result of leakage from the anterior or posterior branch of the middle meningeal artery or the lateral sinus or its tributory veins need not cause any signs of motor deficit, or any other specific signs if they happen to be cerebellar; surgical shock or dehydration will keep the intracranial pressure from rising, and an associated contusion or laceration of the brain will change clear to bloody cerebrospinal fluid. The unreliability of these so-called classical diagnostic signs can be better appreciated when it is realized that the first attack of unconsciousness is caused by the associated brain injury. The conscious interval is present only when the patient has recovered from the brain injury and is not yet rendered unconscious by the growing extradural clot. The final unconsciousness arises from pressure exerted on the brain by the growing clot. It is apparent, therefore, that the conscious interval, which is what makes the history pathognomonic, depends for its presence and length on the severity of the brain injury and the rapidity with which the clot grows to a lethal size. It can be, therefore, and in large numbers of instances is, fleeting or absent, thus making the history no different from that of a severely lacerated brain. Finally, a slow pulse and a rise in blood and pulse pressures are not signs of an extradural hemorrhage any more than changes in the eye grounds are. They, like the latter, are evidences of severe and rapidly rising increased intracranial pressure. Because they usually do not develop until compression and early edema of the medulla is present and because by that time most patients who have an extradural hemorrhage are moribund, they are not often seen in connection with this lesion. Tradition and text book statements would give one to understand that this combination is common and is almost as pathognomonic of extradural hemorrhage as the history. However, this, like the belief held by many surgeons that extradural hemorrhages are exclusively arterial and never venous, evidence lack of experience rather than accuracy of observation.

Extradural hemorrhage should be suspected as a diagnosis whenever its pathognomonic history has been obtained. Patients who do not give a history of the first period of unconsciousness after a blow on the head and are first seen by the surgeon while conscious are only slightly less liable to be suffering from this lesion than those with the pathognomonic history. This will be doubly true if unconsciousness develops later and thus forms two-thirds of the pathognomonic triad. The diagnosis will be confirmed to the extent of necessitating immediate operation without the need of further evidence if a fracture line can be shown by *x*-ray to cross any part of the middle meningeal artery, the superior sagittal or the lateral sinuses. In case of doubt or in case a patient on whom a diagnosis of nonoperable cerebral injury has been made, gets worse or fails to improve in spite of efficient nonoperative therapy, unilateral and, if necessary, bilateral exploratory transtemporal trephinations should be promptly resorted to as a final diagnostic procedure.

Many half truths have been written and innumerable conclusions based on inadequate evidence have been published relative to the diagnosis and symptomatology of subdural hematomas. The profession at large persists in thinking of these clots as a

chronic disease and fails to recognize that every chronic condition is acute at some time and even worse—fails to act on the well recognized doctrine that proper therapy during the acute stages will do away with the chronic feature. For simplification and to eliminate as much as possible the element of personal interpretation on the part of the surgeon, I have classed subdural hematomas as acute when they are diagnosed during the time that the brain injury is still recognizable in terms of pathology, and as chronic when the brain injury is a thing of the past, healed and present only as the final end point of the acute process. I propose to deal only with the diagnosis of the acute subdural hematomas at this time. I have covered other aspects of this problem in a study of 310 such clots which is published elsewhere.[6]

Before dealing with the diagnosis, it should again be emphasized that all subdural hematomas when they are first formed, consist of blood and cerebrospinal fluid mixed in varying proportions and incarcerated in the cerebral or cerebellar subdural space. If the blood is at a maximum and the cerebrospinal fluid at a minimum or absent, a solid clot will be formed. This will be organized by the dura from the surfaces toward the center and if not removed, will eventually become either a fibrous thickening on the dura or a collection of encysted fluid. While such clots are forming and before organization begins they act as expanding lesions and raise the intracranial pressure. If the clot is large and organizes slowly, this pressure may stay elevated for a number of months. If the clot organizes quickly and the brain adjusts its volume easily to this new cranial content, the intracranial pressure may return to normal in a few days. This is the least common form of subdural hematoma and is the one written about as "chronic subdural hematoma." It is the type that is produced by birth injuries and any trauma that ruptures a bridging vein without associated brain injury, or a pial vein with minimal brain injury. The history is one of a trivial head injury accompanied either by fleeting unconsciousness or none at all. There is frequently an interval in which no symptoms are present—the so-called latent period—to be followed when the clot grows big enough, by indefinite subjective complaints such as headache, lack of interest and initiative, impaired judgment and the like or by the usual signs and symptoms of increased intracranial pressure such as are seen with cerebral tumors. The diagnosis can and should be suspected when persistent subjective symptoms or persistently increased intracranial pressure follow an insignificant brain injury and particularly when that injury has been properly diagnosed and adequately treated. The diagnosis will be rendered probable by demonstrating, by means of ventriculography, a space-occupying lesion on the surface of the cerebrum and will be confirmed by the finding of the clot at either an exploratory transtemporal trephination or beneath a flap craniotomy.

The commonest type of subdural hematoma, whether acute or chronic, is the one that is always associated at the start with a contused or lacerated brain. This hematoma is formed from a mixture of blood and cerebrospinal fluid which is incarcerated within the cerebral subdural space. The blood comes from ruptured pial veins and the cerebrospinal fluid from the subarachnoid space. Both reach the subdural space by way of a tear in the arachnoid. Since this tear closes in a few hours, the mixture is henceforth irremovable except by mechanical drainage. Because of the osmotic relationship between this subdural mixture on one side and the normal cerebrospinal fluid on the other side of the arachnoid, which acts as a dialyzing membrane, the hematoma is an expanding lesion for times varying from five weeks to ten days, depending on the amount of blood that has been originally mixed with the cerebrospinal fluid in the subdural space. Thus, because this type of subdural hematoma will always be found in association with either a contused or lacerated

brain, the ordinary signs and symptoms that are present by virtue of the brain injury will have no other or special diagnostic significance in relation to the subdural hematoma. The increased intracranial pressure that is produced by the hematoma will be present anyhow as a result of the brain injury as will changes in the cerebrospinal fluid. They also will not have any special diagnostic significance in relation to the hematoma. The history will be that of a head injury that produced unconsciousness. In a word, it is impossible, except by a good guess, to make a differential diagnosis of the common type of acute cerebral or cerebellar subdural hematoma from the commoner forms of nonoperable brain injuries by the usually available signs and symptoms. Other and more complicated diagnostic procedures must be used. Only the intracranial use of air and the exploratory operation are left. Although advocated by certain authors,[7,8] I do not approve of the use of air in the face of acute cerebral injury and, therefore, have had no experience with it. On the other hand, a considerable experience with diagnostic subtemporal trephinations has satisfied me that if properly done in a properly staffed and equipped hospital, they do not materially add to the patient's risk if negative, and often save his life if positive. They not only establish the diagnosis but institute much needed therapy. Inasmuch as all these patients will or should have a diagnosis of contusion or laceration of the brain made on them promptly and since with the establishment of that diagnosis efficient nonoperable therapy will have been instituted at once, failure on the part of the patient to improve or the fact that he is getting worse in spite of adequate therapy should suggest to the surgeon that his diagnosis is wrong or incomplete and should make it mandatory for him to proceed to further investigation. It is at this point that the exploratory trephination is indicated—not because a diagnosis of subdural hematoma has been made but because the only diagnosis justified by the evidence collectible to date has not led to proper therapy, and because an unrecognized subdural hematoma is a strong possibility as a cause of this failure. I am convinced that no one can diagnose an acute subdural hematoma prior to exploratory trephination except as a guess.

Intracortical hematomas are not common as the result of a blow on the head. They cause no characteristic signs or symptoms which will serve to differentiate them from the more serious types of nonoperable cerebral pathology. They have no typical history and just as with the acute subdural hematomas cannot be diagnosed by any means short of exploratory trephination except by guesswork.

COMPLICATIONS

Of the complications that develop in association with acute craniocerebral injuries, the most important are surgical shock and toxic dehydration. Others that are less important are meningitis, aerocele and arteriovenous aneurysm.

Surgical Shock. The diagnosis of surgical shock need not be gone into in detail here. Suffice it to say that from a practical point of view any patient who has a pulse pressure below 20 mm. of mercury can be considered to be in surgical shock and should be dealt with accordingly. On the other hand, any patient whose pulse pressure is over 20 mm. of mercury can be considered as not being in shock. Those whose pulse pressure is exactly 20 mm. must be regarded as borderline cases whose final classification must await further observations. Surgical shock will produce of itself an intracranial pressure that is well below the expected minimal level for the associated cerebral injury.

Dehydration. Dehydration as a cause of symptoms and as a significant complication of acute craniocerebral injuries is usually regarded as a myth. Criteria that demand exact diagnoses based upon demonstrable undisputable facts rather than diagnosis by inference will, however, if the surgeon's

experience amounts to anything, shortly demonstrate that this comfortable theory has no basis in fact. Unconscious patients who, after injury, have been exposed to the sun following their accident; who have not been given sufficient fluids because of their inability to ask for them, or because of arbitrary limitation of fluid intake, or who have been vomiting; or who, in spite of having a high temperature have been surrounded by artificial heat in the shock-room of the hospital; or have been treated with artificial dehydration injudiciously and, as far as the condition goes, ignorantly, are all potential sufferers from toxic as opposed to therapeutic dehydration. The diagnosis should be suspected in the face of such a history—particularly if the patient's skin is dry, if he has developed a fever, or has been having, if an adult, less than 5,000 cc. of fluid every twenty-four hours regardless of the other methods of treatment. The diagnosis is probable if, in addition to any of the above, the patient who has previously been getting better suddenly begins to get worse and again develops signs and symptoms that he had had previously but that had disappeared under treatment. The diagnosis is certain if it can be demonstrated that in addition to the above his intracranial pressure is below the expected minimal level that is appropriate to the cerebral pathology that is present. In severe cases and in children it can be further confirmed by the demonstration of acetone or diacetic acid in the urine or an abnormality of the carbon-dioxide combining power of the blood, in which cases it leaves the group of toxic dehydration and becomes acidosis.

Meningitis and so forth. The diagnosis of meningitis is made from the classical signs and presents no problem other than that associated with identification of the infecting organism and the route by which that organism has reached the meningeal spaces. This has been dealt with sufficiently above, as has the diagnosis of an arteriovenous fistula. Aeroceles are diagnosed by *x*-ray.

CONCLUSIONS

A diagnosis should be a word picture of the pathological condition that is present and that leads to the need for making a diagnosis. Its accuracy will vary directly with the objectivity of the signs and symptoms upon which it is based, and their freedom from the possibility of individual variations of interpretation by the surgeon and the patient. This holds particularly true in the diagnosis of acute craniocerebral injuries.

A prerequisite to the diagnosis of any acute craniocerebral injury, except perforating wounds of the skull, is the demonstration of the fact that there has actually been an injury to the patient's head and that he has been knocked unconscious as the direct result of that injury.

Accurate diagnoses of uncomplicated nonoperable craniocerebral injuries are dependent first upon the history as noted above, then on a study of the make-up and pressure of the cerebrospinal fluid. Diagnoses made without these data are inferential rather than factual and can be successfully disputed before impartial judges.

Accurate diagnoses of uncomplicated operable craniocerebral injuries will always include a word picture of the associated nonoperable cerebral condition. In addition, certain data that are obtainable only by special *x*-ray examination, palpation through a scalp wound, a detailed history of the development of symptoms after the accident, and the use of an exploratory trephination whenever the patient's progress does not coincide with the diagnosis already made are necessary.

More attention should be paid to the possibility of significant complications of acute craniocerebral injuries. This is particularly true in regard to surgical shock, toxic dehydration and acidosis.

Accurate diagnoses of common complications of acute craniocerebral injuries will depend upon a knowledge of the cerebral pathological condition that has been produced by the head injury, a study of the pulse pressure, a study of the patient's

history in relation to exposure to heat, fluid intake and limitation, the administration of dehydrants and the level of the intracranial pressure.

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This (neuralgia) is a painful symptom attributed to a nerve or its branches. The pain has a "pricking" quality, often of exquisite intensity, intermittent in duration (free intervals), and may have a wide distribution or radiation. It is an expression of a lesion involving nerve roots, nerve trunks, or their ganglia.

STAB WOUNDS OF CHEST WALL AND LUNGS

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THE concentration of thousands of men at various places in the country and particularly in the South, is likely to increase the incidence of stab wounds seen by both military and civilian physicians. A review of the subject is timely.

No class of wounds, unless it be in a few centers of the brain, involves the essential physiologic processes of the body more rapidly than wounds of the chest. In addition to the rapid physiological changes which must be met, many pathologic sequellae may be expected. No class of wounds requires more accurate judgment at onset or more persistent care during its course.

Injuries and wounds of the chest have been classified in several ways. Lilienthal[1] divides them into superficial, penetrating and perforating, the last two classes being differentiated only by degree. A simpler classification is given by Cole[2] into external and internal chest injuries, while Bigger[3] calls them perforating and nonperforating. A similarly explicit description may be made of superficial wounds which involve layers of the thoracic wall to the parietal pleura, and deep wounds which extend through the parietal pleura to varying depths.

An analysis of the type of wound upon which this paper is based may be estimated from the figures for the thoracic service in a two hundred sixty bed city and county hospital for one year and will serve as an average cross section of these injuries. The wounds are similar to those discussed by Elkin.[4] Two hundred ninety-seven patients with chest wounds were received in the emergency room. General figures showed eighty-eight to be white and two hundred nine to be colored; two hundred four were males and seventy-one females; twenty-two were dead on arrival. The causative agents of the chest wounds were; nineteen from automobile accidents, eighteen from gunshot wounds, seven from industrial injuries or minor accidents about the home or farm. The remainder of the two hundred fifty-three wounds were hand inflicted apparently with homicidal intent. It is with this last group that we are interested. Of these chest injuries, ninety-four were committed with knives, fifty-seven with ice picks, twenty-one with razors, one with a hatchet and in fifty-eight cases the patient either did not know or was unwilling to tell what weapon was used. These figures are summarized in Figure 1. Fortunately for the hospital, the majority of the chest wounds are superficial in type, and of two hundred fifty-three stab wound cases, only one hundred four were admitted to the wards.

Among the negroes, (the two to one offenders) the ice pick is a favorite weapon; it costs five cents, is not conspicuous, looks bad when drawn and with a metal handle may easily be thrown a hundred or more feet when it is time for flight. The "switch" or spring released knife, the blade of which varies from four to six inches, is a more expensive favorite. A "frog" is a single bladed knife, three and one-half to five inches, whose pin is oiled and spring filed so that it can often be opened "on the throw" almost as rapidly as a switch. With such social accoutrement, negro night life adds to emergency room excitement.

Peculiarly, very few bayonet wounds of the chest were seen in the hospitals during the last war. The reasons given for this are: first, that the wounds were so severe that the victim failed to survive the trip, second, that the armies were taught to

lunge for the abdomen so that the bayonet would not be caught in the ribs and third, in case the bayonet was difficult to withdraw to shoot it loose. During the Civil War, with much hand-to-hand fighting, there were reported in the Federal Records by Barnes[5] only thirty-eight sabre and bayonet wounds in a total of 246,712 wounded.

	Total	White	Colored	
Automobile	19	11	8	
Gunshot	18	13	5	
Industrial or Accident	7	5	2	
Hand Inflicted				Mortality
Dead on arrival	22	4	18	
Knife	94	40	54	
Ice pick	57	9	48	8 or 3.5% and 15% for the admitted cases.
Razor	21	3	18	
Hatchet	1		1	
Unknown	58	3	55	
	297	88	209	

Fig. 1. Table of chest and stab wounds for one year.

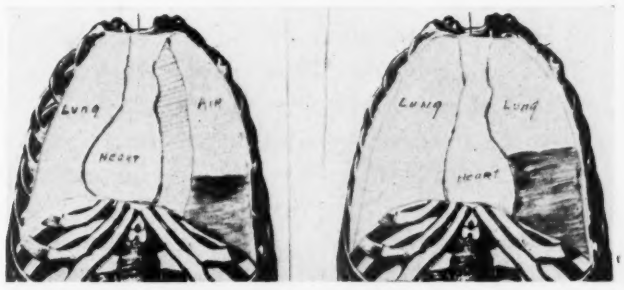
Superficial chest wounds, may involve only the skin and fascia or may extend through the muscles, ribs, and intercostal muscles and vessels to the parietal pleura. Not only do these wounds deserve careful examination, but a description of the injuring force or weapon and the direction of the force should be ascertained. This history is frequently unobtainable because of shock or fright, or both to the patient and the irresponsible statements of excited witnesses. The surgeon must work cautiously, unassisted by a history, to determine the extent of the wound.

The general condition of the patient and the physical findings in the chest may be an aid or delusion. It is often difficult to determine whether the rapid pulse is due to hemorrhage before arrival at the hospital, shock from an internal pneumothorax or the excitement of having been slashed by an irate husband or jealous wife. Morphine and reassurance may quickly cure what appeared to be an exsanguinated patient, and an adequate pulse pressure confirm the cure. In the examination of the chest, it must be remembered that a peripheral injury will cause a degree of splinting commensurate with the pain, and decreased expansion and respiratory sounds. Also, a definite atelectasis of a portion of the lung may be produced by an external blow.

The superficial wound should be carefully examined and, contrary to some authors (Head,[6] Davis,[7] and Everett[8]), explored, débrided and accurate hemostasis established. More care than is usual in soft tissue wounds should be used. The attendant dangers of any soft tissue wound—hematomas and infection—are present, but the danger of a hematoma or infection following a small opening inward through the constantly moving thoracic wall is the reason for most meticulous surgery. The usual patient with a stab wound has a dirty body and clothing and requires careful débridement.

The external bleeding from intercostal vessels is particularly disturbing and must be stopped. Instances of extrapleural but intrathoracic hematoma with marked exsanguination have been reported. The intercostal vessels themselves are usually well protected, particularly in the adult's lower chest, by lying in a groove on the inferior and inner aspect of the ribs. If the bleeding point is not seen by simply spreading the wound edges, strong retrac-

tion of the intercostal and the serratus muscles may bring the severed ends of the vessels into view. Should this fail, a sub-pleural suture may be passed around the entire rib above and below the site of the bleeding. As a last resort, a section of the rib must be removed without hesitation.



A B

Fig. 2. A, diagram of hemopneumothorax with compression of lung and displacement of heart from interpleural air and basal collection of blood. B, diagram of intrapulmonary type of bleeding with little heart displacement.

After closing the superficial wound, it is well to strap that region of the chest thoroughly. The strapping and immobilization are done for the patient's comfort and in an effort to prevent delayed bleeding, which is aided by the rhythmic movements of the chest.

Because many of these wounds are contracted in tetanus infected localities and are often puncture wounds in nature, the use of tetanus antitoxin should be made routine.

Deep wounds extend through the parietal pleural and the difficulties manifoldly increase. Should the external pleura alone be injured, there is the possibility of a pneumothorax. The amount of air will depend on the size of the opening through layers—the sucking wound. With the sucking in of air, blood from the external layers of the wound will also be drawn into the pleural space producing a hemopneumothorax. If the wound extends to the lung tissue, we have the possibility of both an internal pneumothorax and an external hemopneumothorax. (Fig. 2A.)

With the formation of the pneumothorax there is collapse of the lung. The extent of collapse will vary with the size of the openings and the presence of pleural adhesions. Graham (quoted by Bigger), has shown experimentally that if the size of the external opening equals the cross section of the bronchus, there will be an adjustment of pressure. However, the soft tissue may make a trap door which closes during expiration and the internal pressure will rapidly increase. This is particularly true when the lung is torn and the patient is coughing. The intrathoracic pressure of the cough is estimated to be as high as 87 mm. of mercury, by Starling,[9] and under such pressure considerable air is forced through even a small lung opening into the pleural space. Should adhesions attach the lung to the chest wall, these may be torn loose, giving a second internal opening or a secondary hemorrhage.

The rapid collapse of the lung and its compression against the mediastinum causes more or less cardiac displacement. The degree of displacement is modified by previous adhesions of the lung to the chest wall and mediastinal fixation. If there be little heart displacement, the compression of the lung against the thin veins and auricles may cause circulatory embarrassment. When the mediastinum is entirely without fixation, the heart may be markedly displaced and swing laterally from the great vessels with each respiration—a condition long recognized in thoracic work as pendulum shock.

Not only is the heart subjected to displacement and embarrassment, but in the presence of extensive pneumothorax, the

remaining lung is frequently robbed of an appreciable portion of its air. With the shallow respiration resulting from shock and pain, a portion of the air in the active lung is shifted back and forth into the dead space of the opposite bronchial tree. The Germans have called this phenomenon "pendle luft," and these patients may actually die from lack of oxygen.

The first treatment in deep wounds is to stop the sucking in of air. This may be accomplished by a few sutures, or if a large wound, by a temporary rubber dam placed over the opening to act as an externally discharging valve. The wound should be repaired as quickly as the patient's condition will permit, under an anesthesia during which a positive pressure may be maintained.

To prepare for surgery, the shock in these patients must be combatted. Morphine, heat and aspiration of the pneumothorax by a needle or valve are the best procedures. Intravenous solutions must be used with the greatest caution. This fact has been emphasized in complications following thoracoplasties, but has not been sufficiently applied to traumatic emergencies. If the patient is suffering from systemic shock, incident to trauma in which the pneumothorax is a minor part, intravenous support of the circulation may and should be used. If the condition is the result of cardiac embarrassment from lung compression and mediastinal displacement, increasing the burden on an already embarrassed right heart may result tragically. Intravenous solutions enter the right heart first, right ventricular pressure at best is only 15 to 20 mm. of mercury, and in the presence of traumatic pneumothorax the veins and right auricle are often struggling against air pressure greater than they can produce.

The immediate needs for severe pneumothorax are oxygen, which at times is supplied by the longer, deeper respirations following moderate doses of morphine, or directly by oxygen with a nasal tube, and a release of the collapsed lung from the high intrapleural pressure. The pleural pressure may be released through an intercostal needle, any of the metal valves designed for this purpose, or a tube, inserted intercostally, with a slit finger cot tied over the end acting as a valve. The finger cot valve is less cumbersome than an outlet under water although the suction with the tube under water may be increased by lowering the receptacle. Of the three methods, the catheter inserted intercostally through a trocar and a slit finger cot tied about the distal end, allows a rapid release of pressure in the emergency room and its action can be easily observed.

Should the pneumothorax be complicated by bleeding, the fundamental compression of the lung remains the same but the treatment must be more active. Bleeding into the pleural cavity may be from the thoracic wall inward or from the lung or both. If from the thoracic wall, the intercostal vessels are the great offenders and must be ligated as soon as the patient's condition allows. Should the bleeding be from the lung, there are two sources, the bronchial arteries or the pulmonary vessels. Little attention in traumatic chest condition has been given the dual blood supply of the lung, although Bettman[10] has called attention to the pathological differences in the two circulations. Should the periphery of the lung and the pulmonary vessels alone be involved, there will usually be found a moderate amount of blood and a moderate pneumothorax. Both of these tend to compress the resilient lung tissue, to close the opening and to check the hemorrhage. The physical findings of hyperresonance, basal flatness and diminished or absent breath sounds are typical of the condition.

Because of the original low pressure of the pulmonary vessels and their early branching, the reduction to capillary pressure is rapid. The increased pressure of the pneumothorax will cause hemostasis in the moderate internal type of wound. If there is no evidence of cardiac embarrassment or signs of increasing hemorrhage,

the patient may be treated generally until about seventy-two hours has elapsed. This length of time allows small vessels to clot and gives an idea of the presence of primary contamination. An estimation of the amount of blood is attempted from the *x*-ray. In reviewing the films, it is well to remember that blood has a tendency to spread over the pleura and give an exaggerated picture (Wood and Eloesser[11]). Also, the pleura rapidly dilutes the blood with serous exudate to increase the fluid as compared with the first picture.

Following war experience, Morelli and Lilienthal advocated aspirating blood from the pleural cavity and replacing it with air. The method was met with considerable opposition by those of the profession who feared infection. While a portion (about 8 per cent) of all hemothorax cases will become infected, the majority of observers (Sonte,[12] Boland,[13] Sandison,[14] and Miller[15]) advocate aspiration and air replacement because: (1) series of cases fail to show a higher incidence of infection, (2) hospitalization is shortened, and (3) there is less residual pleural thickening and atelectasis of the lung tissue.

Later complications of lung injuries are empyema and abscesses with their long attendant difficulties, and acute respiratory infection. These must be treated as the case demands.

When a bronchial artery of medium size is severed, a very serious problem is presented. The bronchial arteries arising from the aorta receive the systemic pressure of the left ventricle. They accompany the main bronchi and their injury usually involves the bronchus. In these cases the bleeding is intrapulmonary. Gurgling, bronchial râles at first heard below the level of the wound are rapidly silenced as the alvoelae are filled with blood. The percussion note is flat. There is usually little cardiac embarrassment or displacement. As the level of the blood rises into the bronchi immediately above the injury, coughing produces small amounts of bright blood. Aspiration usually obtains only a few cc. of blood. However, should the rent in the peripheral lung tissue be large, the blood may find its way directly into the pleural cavity. In such a case aspiration of the blood and compression pneumothorax with air may be utilized. The patient is in no condition for exploration or lobectomy. When exsanguination is eminent and donors not available, autotransfusion of the aspirated blood directly into the median vein may be used. A 50 cc. syringe with an outlet stop cock, glass connections and rubber tubing make a satisfactory apparatus. The side opening of the stop cock is utilized for transfusion and air replacement.

The chest wound with bronchial artery bleeding and bronchial damage is the most difficult type and the least satisfactorily handled. With the lung tissue filled with blood, interpleural pressure with pneumothorax has little effect on the solidly filled organ as it is impossible to inject sufficient air pressure to stop any except a small bronchial vessel. Intravenous therapy will increase the blood pressure and the bleeding. Exploration for the bleeding vessel is impractical. Phrenicotomy, with the weight of the blood on the diaphragm, may cause its descent and increase the intrapleural area. Supportive measures and allowing the rise of the blood level within the lung tissue to compress the vessel is the best procedure.

If the bronchial vessel is small and only part of a lung involved, the patient may respond to the treatment of "masterful inactivity." Very slow resolution of the blood with a low (100°), constant temperature for about eight days may result. Six months later a narrowed atelatatic shadow, slight diaphragm rise and emphyzema of the lung above may be the only residua.

Should the intrapulmonary bleeding be massive (approximately two-thirds of a lung), recovery from the bleeding will be followed by a disappointing series of complications. A gangrene of the alveolar tissue results in a sepsis. This is usually

discernible at about the fifth to sixth day by a septic temperature, and leucocyte rise. Drainage, closed or open, has no appreciable results. Radical rib resection, removing large quantities of gangrenous material and packing and irrigating with various antiseptics, produce no results other than a bronchial fistula and the patient grows slowly more septic. For these reasons, I advocate lobectomy in the intrapulmonary type of bleeding at the first signs of sepsis. A diagram of the simple hemopneumothorax as compared with the intrapulmonary type of bleeding is seen in Figure 2.

CONCLUSIONS

1. Care must be taken in examining and repairing superficial chest wounds.

2. The immediate closure of deep chest wounds should be done and aspiration or valve control of internal pressure should be used.

3. The mistaking of systemic shock for right heart embarrassment and the danger in using intravenous solutions in the presence of increased intrapleural pressure is emphasized.

4. There is a difference in treatment and results of wounds involving the pulmonary and those involving the bronchial vessels.

5. Lobectomy or pneumectomy should be used in massive intrapulmonary hemorrhage at the first signs of sepsis.

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DIAGNOSIS IN ABDOMINAL TRAUMA*

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WHEN a traumatic lesion of the abdomen, due either to penetration or blunt violence, is suspected or known to exist, diagnosis embraces more than the mere determination of whether peritoneal perforation has occurred, and even more than estimation of the probable existence and extent of visceral injury. Upon a complete determination of the patient's status in respect to shock, hemorrhage, and associated injuries, depend both the selection and the timing of the therapeutic measures which have been discussed elsewhere.[1,2] In addition to immediate or preoperative studies, observations at the time of operation, and recognition of the late complications which follow traumatic lesions of the abdomen, are still other important phases of diagnosis.

An account of the circumstances under which the injury was incurred, as well as a description of the mechanism of the injury, often furnish valuable information. (Fig. 1.) For instance, in considering the possible damage done by a blunt force or by a projectile following a known course, if the urinary bladder was recently emptied it may escape injury; while if distended at the time the wound was sustained, the bladder may not only have been struck but is likely to have been extensively lacerated as a result of the explosive force exerted by its noncompressible fluid contents. (Fig. 2.) Likewise, evacuation of the bowel shortly before injury favorably influences the amount of spillage of intestinal contents. The physical attitude of the patient when wounded may greatly influence the course of a projectile, and in the instance of subcutaneous wounds, the type and direction of the blunt force often suggests the probable location of visceral lesions. Information concerning the period of time elapsed since the injury was sustained, and the character of first aid or other previous treatment is also of value in planning the management of patients with abdominal injuries.

Prompt determination of the existence of abdominal visceral injury, extensive hemorrhage, shock, or associated chest as well as craniocerebral injury is especially necessary when patients are to be transferred in airplanes flying at high altitudes. Lovelace[3] has reported on the deleterious effects caused by reducing atmospheric pressure in the presence of any of these conditions, and has also drawn attention to the increased outpouring of stomach and intestinal contents caused by expansion of gas in these viscera when atmospheric pressure is lowered. Only those ambulance air transports which have cabins that can be sealed to permit maintenance of barometric pressure near that at ground level are safe conveyances for seriously wounded patients.

SYMPTOMS

Pain. Pain which follows traumatic lesions of the abdomen is not constantly and directly related to the existence, location or extent of visceral injury. Usually experienced immediately, its onset may be delayed for several minutes, or even several hours, either because of excitement at the time of the injury, or, more often, because pain following abdominal injuries is not due so much to visceral damage as it is to peritoneal irritation caused by blood or the spilled contents of hollow viscera. The extravasation of acid stomach juice may

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immediately cause sudden severe, diffuse pain, while perforation of the cecum, with spillage of only a small amount of solid or dependable symptom. Trauma to the lumbar plexus sometimes causes pain in the corresponding lower extremity, so that

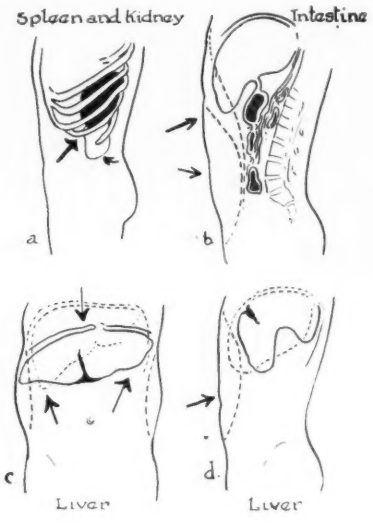


FIG. 1.

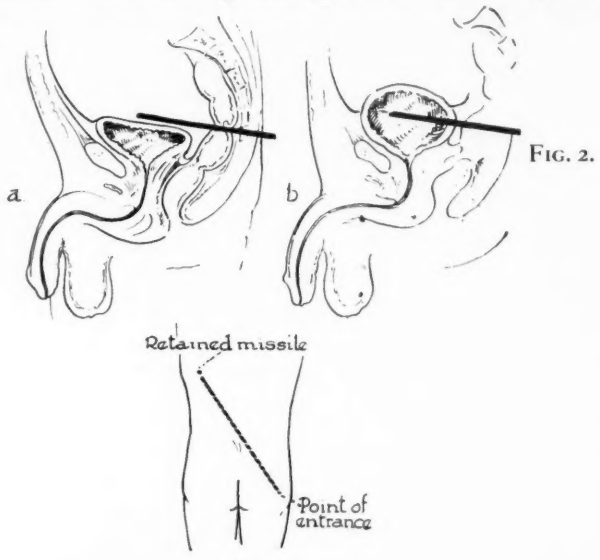


FIG. 2.

FIG. 3.

FIG. 1. Diagrams illustrating the mechanism of subparietal injuries to abdominal viscera due to the blunt forces indicated by arrows. The fixation, location, size, and turgor of the liver and, to a lesser degree, of the spleen and kidneys make them particularly susceptible to injury. Fixation by a mesentery or by a vascular pedicle is an important element in the mechanism of injuries to hollow viscera which are caused by blunt violence. In addition to the compression injuries caused by motor vehicle steering wheels or by intact ribs, broken steering wheels and fractured ribs may cause penetrating wounds.

FIG. 2. Diagrammatic illustration showing how, depending on whether it is empty or filled, the bladder may or may not escape injury by a missile traversing a given track. When a projectile strikes a distended bladder an "explosive" or "bursting" type of injury may be produced as a result of the incompressibility of the fluid bladder contents.

FIG. 3. Diagram indicating the intraperitoneal course of a missile which has entered at a point remote from the abdominal wall. Abdominal viscera are frequently injured by missiles which enter through the buttocks, perineum, thigh and thorax.

semisolid material, may be followed by practically no discomfort, especially if there is no associated intraperitoneal hemorrhage. Soldiers with extensive wounds and even with evisceration of abdominal organs have often continued in action, and both soldiers and civilians with such injuries may walk considerable distances after being wounded.

Pain following injuries limited to the abdominal wall may be greater than that which sometimes exists when the peritoneum has been penetrated. A localized painful area distant from the point of entrance may, when there is no wound of exit, indicate the region in which the missile is lodged, but this is a rare and unthe injured individual may consider that his wound primarily involves the thigh or leg rather than the abdomen. When large objects bluntly injure the abdomen, the sensation experienced is similar to that produced by any violent blow. Small projectiles can cause a sensation as if a blow had been sustained by a large object, but the sensation usually produced by them is sharp or stabbing.

Pain may be either transient or persistent, and may be accompanied by collapse. When intestinal evisceration has occurred, there may be cramping pain due to peristalsis; but this is often not the case, because of the usually rapidly developing adynamic ileus which involves the extruded

loop as well as that portion of the bowel which has remained within the abdomen. Pain referred to the root of the neck may be due to intraperitoneal hemorrhage or to the subdiaphragmatic accumulation of gas which has escaped through a perforation of the stomach. There is ordinarily not sufficient gas either in the small or the large intestine to produce this symptom.

Injuries in other parts of the body sometimes so completely direct attention to these parts that the pain due to the abdominal injury is overlooked. Whether the patient has or has not received morphine must also be taken into consideration in assaying pain.

Nausea and Vomiting. The presence or absence of nausea and vomiting does not indicate whether or not intraperitoneal injury has occurred. The degree of these symptoms may be modified by the injured person's normal tendency to become nauseated and to vomit. These symptoms may follow injury to practically any abdominal area or viscus, particularly the stomach, esophagus and kidneys, but they may be absent, regardless of the location or character of the wound, the amount of spillage of alimentary tract content, or the extent of either intraperitoneal or extraperitoneal hemorrhage. Contrary to what might be expected, hematemesis often does not accompany stomach injuries.

Miscellaneous Symptoms. Air hunger is usually experienced by patients who have suffered considerable blood loss, and when hemorrhage has been extensive, air hunger may even be evident to the examiner. Dyspnea, most often observed following abdominothoracic injuries, may be pronounced even though no chest injury exists.

Bladder or rectal tenesmus may follow urinary bladder or rectal wounds, respectively, and one or more bowel movements sometimes occur shortly after the injury. The material excreted by bowel may contain or be coated with blood, but early spontaneous hemostasis often prevents the appearance of gross amounts of blood, and in any event, the passage of blood by rectum is rarely observed by the injured person. Varying degrees of hematuria may be noted by the patient, but the suppression of urine which frequently follows injuries to the urinary tract may cause considerable delay in the appearance of hematuria.

PHYSICAL EXAMINATION

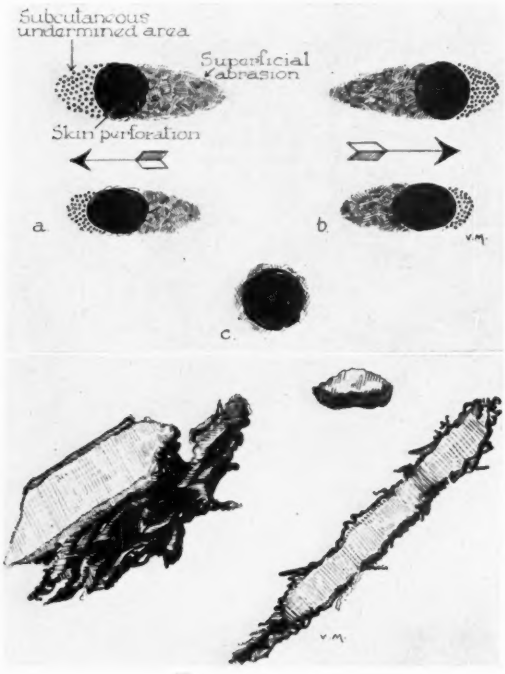
A general physical examination, even quickly performed, can not only reveal injuries unrelated to possible abdominal trauma, but permits detection of wounds produced by missiles which enter the abdomen via the buttocks, thighs, perineum or thorax. (Fig. 3.)

INSPECTION

General Appearance. Patients with abdominal wounds usually seem ill in proportion to the extent of their injuries, but this relationship is influenced by the time which has elapsed since injury, by variations in reaction to an equal degree of pain, and by the amount of associated shock and hemorrhage. A calm facial expression, perfect composure and animated interest in the surroundings may reflect a phlegmatic disposition and can be misleading. The appearance of the patient is also affected by such first aid attention as the administration of morphine and the application of external heat. There may be extreme restlessness or excitement, but more frequently, the patient is either quiet, drowsy or even apathetic. Especially in the instance of soldiers, because of loss of sleep or sustained physical and psychic tension, a desire to secure rest and to sleep may supercede any concern about the wound. The color may be good, or there may be some pallor, and occasionally even cyanosis, especially if there is also a chest injury. Perspiration is frequently profuse. Dyspnea and rapid respiration are usually present when there is an accompanying thoracic lesion. Air hunger may be detectable by inspection.

The abdomen usually reveals a diminution or absence of the movement which

normally occurs synchronously with respiration. Although breathing which is either entirely or principally thoracic in



FIGS. 4 AND 5.

FIG. 4. Above. Drawing illustrating various types of external wounds in relation to the courses taken by bullets. A concentric area of abrasion and contusion indicates that the bullet has taken a straight course. If the area of abrasion is to the right of the edge of the wound, it signifies that the missile has passed from right to left, and vice versa if the area of abrasion is to the left of the edge of the wound. Furthermore, the amount of undermining beneath the edge of the wound opposite the area of abrasion may also indicate the obliquity of the course of the missile.

FIG. 5. Below. Fragments of high explosive shells used in the present European War. Various-sized fragments of this sort, along with bullets, are responsible for most of the wounds in contemporary warfare. Because of their irregular shape, fragments of high explosive shells cause ragged lacerations, and the larger fragments are especially likely to be retained if traveling at low velocity.

type may be observed when the injury is limited to the abdominal wall, the absence of abdominal excursion suggests that there is intraperitoneal extension of the wound.

A bluish area may occasionally be seen in the region of the umbilicus when there is a large amount of blood in the peritoneal cavity. Inspection of the abdominal parietes anteriorly, laterally and posteriorly, in addition to furnishing information concerning the wounds of entrance or exit, may also reveal external bleeding, or the discharge of urine or feces. A plug of omentum in or protruding through a wound at once reveals that the peritoneum has been perforated. A knuckle or loop of either intact or injured intestine, or a portion of the stomach, spleen or liver sometimes is found projecting into or completely through the parietal wall wound. Occasionally, a bullet or shell fragment can be seen in an open wound, and projectiles lying beneath the skin often can be detected by the bulging of the skin which they cause, especially when surrounded by a hematoma. Subcutaneous rupture of an abdominal wall muscle may be indicated by a hematoma at the site of injury.

Bulging of the flank, caused by outpouring of blood and intestinal content, may be evident. Abdominal distention and a corrugated appearance produced by distended intestine are observed only after the onset of ileus or peritonitis. When the spinal cord is injured, distention of the urinary bladder may be detected.

Study of entrance and exit points or even of an entrance wound alone can help in determining whether peritoneal penetration has occurred.[4] The entrance wound produced by a bullet is almost invariably smaller than the caliber of the bullet, while the wound of exit is usually larger than the caliber of the bullet and these wounds are frequently irregular. A concentric area of abrasion and contusion surrounding the point of entrance indicates that the bullet has taken a straight course, and that viscera in the region have probably been injured. A superficial abraded area to the side of the wound signifies that the missile has approached from that side. Obliquely directed bullets undermine the skin opposite the area of surface abrasion and, since the areas of contusion and undermining are increased with the obliquity, the more superficial the bullet tract, the larger the superficial contusion and the

greater the undermining. (Fig. 4.) These observations are important in estimating the extent and direction of superficial, non-

ularly grave sign is semierection of the penis, which is reported to be always followed by death.

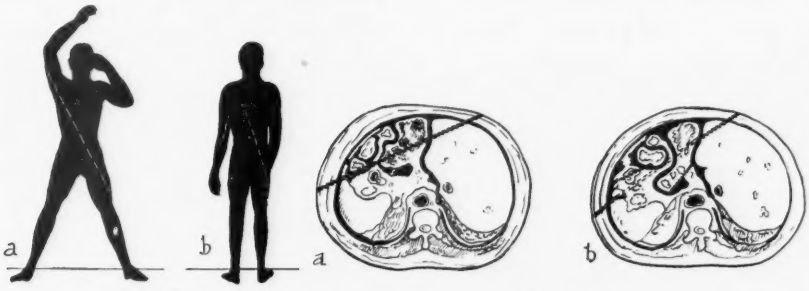


FIG. 6. FIG. 7.

FIG. 6. Diagrams indicating how variation in the relative position of parts of the body may make it appear that a missile has taken an extremely erratic course. (*a*) Indicates the position of the patient at the time of injury; (*b*) indicates how an impression of a bizarre course of the missile might be gained from studying the wound track while the patient is lying on an operating table.

FIG. 7. Diagrammatic representation of the shift in the relative position of abdominal viscera and wound tracks caused by a change of posture or by respiration.

penetrating bullet wounds of the abdomen, which cause pain and at times nausea, vomiting, tenderness and rigidity. Wounds produced by the irregular or jagged projectiles responsible for many of the injuries in modern warfare (Fig. 5), yield less definite information than can be obtained when wounds are caused by bullets.

The apparently bizarre course of projectiles frequently ascribed to deflection or ricocheting is often really due to the patient's bent over, crouched, or partially turned position at the time of the injury. Difficulty in accounting for the location of some visceral wounds found when the patient is lying on an operating table may be due to a shift in the position of organs from that which existed at the time of injury. (Fig. 6.) Furthermore, a shift in the relative position of viscera after injury has occurred, and during various phases of respiration sometimes explains the apparently erratic courses of missiles. (Fig. 7.)

In the condition termed muerte subperitoneal (subperitoneal death) described by Bastos,[5] there are premonitory symptoms of coldness of the extremities, cold sweats, cyanosis, feeble and rapid pulse, but no accompanying signs of peritonitis. A partic-

PALPATION

Palpation of the Skin. Except when there is elevation of temperature due to peritonitis or wound infection, the skin is usually moist and cool. Subnormal surface temperature is frequently accompanied by subnormal oral and rectal temperatures.

Pulse. The pulse is frequently rapid and thready, but a slow pulse does not necessarily indicate that serious abdominal injury is not present. Bradycardia may occasionally result from the absorption of bile which has been extravasated into the peritoneal cavity following liver or biliary tract injuries. Visceral perforation, particularly when involving hollow viscera, and if unassociated with hemorrhage, shock, or infection, may not cause an increase in rate or impairment of quality of the pulse. The prognosis is usually good when the pulse rate does not exceed 100 beats per minute, while many patients with a pulse of 120 or higher die. A rapid pulse when the patient is first seen may not be entirely due to shock and hemorrhage, since pain or excitement may contribute to causing the acceleration. Considerable slowing of the pulse often follows even a short period of rest and quiet. It has been suggested that a persistently rapid pulse or an increasing

pulse rate are indications for operation, but changes in the pulse are frequently a relatively late indication of the existence and progress of shock and hemorrhage.

Tenderness. Evaluation of abdominal tenderness is often difficult. Tenderness due to a wound of the abdominal wall is ordinarily localized, while tenderness due to visceral injury and extravasation of blood or fluid gastrointestinal contents is usually diffuse. If only a small amount of solid or semisolid feces spills into the peritoneal cavity, there may be little or no tenderness. A tender tract may be sometimes traced, corresponding to the course of the missile in the abdominal wall, and an area of localized or circumscribed tenderness may indicate the resting point of a missile in the abdominal wall or within the abdomen. Tenderness is sometimes due to distention of the urinary bladder.

Rigidity. The degree and location of rigidity can lead to an incorrect estimation of the patient's condition in respect to abdominal penetration or visceral injury. Abdominal rigidity frequently accompanies low thoracic injuries, and is usually associated with wounds of the abdominal wall, the pelvic floor and even of the thigh. The amount of blood or urine, as well as the character and volume of spilled gastrointestinal contents determine the degree and extent of rigidity. Extravasation of solid intestinal contents may be followed by undetectable rigidity, while even a small amount of acid gastric juice is likely to cause diffuse board-like rigidity.

The following observations concerning rigidity in abdominal casualty cases have been recorded by Wallace:[6] (1) Rigidity may be slight and may be either general or local. (2) It may be confined to one part or it may become diffuse, generally indicating an intestinal lesion. (3) There may be alternating presence and absence of rigidity, a finding sometimes accompanied by extensive injuries. (4) The term "apprehensive rigidity" has been applied to the condition in which the abdomen looks soft and moves on respiration, but hardens when the hand approaches to palpate. As a rule, it is accompanied by no serious injury. (5) Board-like rigidity, when accompanied by extreme pain, nearly always indicates that there is a wound of the gastrointestinal tract. (6) Complete absence of rigidity is often a very bad sign, although a lax abdomen may exist with hollow visceral lesions and the patient be in good state. (7) Morphia in moderate doses seems to have very little effect on the state of the abdominal wall.

Palpation of the abdominal wall, the lower chest wall, the buttocks and the upper parts of the thighs may reveal a missile lodged superficially either near the entrance wound, or in an area where a wound of exit would have occurred had the projectile travelled slightly farther. Subcutaneous rupture of the abdominal wall caused by blunt force may be revealed by palpation of a gap in a muscle, usually with a surrounding hematoma. The detection of crepitation may, in early cases, be due to subcutaneous emphysema following a thoracic wound, while crepitus in late cases may be due to gas bacillus infection. Occasionally, the diffusion of air trapped beneath a flap of soft tissue, as well as the diffusion into the parietal wall of gas which has escaped through a stomach perforation, is responsible for crepitation in the abdominal wall, and this sign may also occasionally be detectable over an abdominal wall hematoma. Ballottement indicates the accumulation of the peritoneal cavity of blood, gastrointestinal contents, or in late cases, fluid inflammatory exudate. Rectal examination is an important part of the physical examination and may reveal injury to the colon, rectum or anus. Rectal examination is especially important when the wound of entrance is located in the gluteal, sacral or perineal region. Not only may a perforation of the anorectal region be felt, but blood on the gloved finger will disclose bleeding from a wound of the colon even above the reach of the examining finger.

Percussion. Absence or diminution of relative or absolute liver dullness may be

caused by free gas in the peritoneal cavity. The absence of this finding even though a gas-filled hollow viscus has been perforated, is sometimes due to the external escape of gas through the abdominal wall wound, while in other instances, the amount of gas which escaped is insufficient to produce the sign. In the presence of peritonitis, gas in the peritoneal cavity may be due to the activity of gas producing bacteria. Percussion may also reveal abnormal dullness in the flank, or shifting dullness due to the presence of blood or other fluid in the peritoneal cavity.

Auscultation. There is seldom an occasion of auscultate the abdomen of patients with recent abdominal injuries. In late cases, however, the so-called "silent abdomen" may indicate the onset of ileus or peritonitis.

Temperature. Oral, rectal or axillary temperature is often subnormal because of shock and hemorrhage and is sometimes as much as two degrees below normal. Elevation of temperature usually indicates peritonitis or other infection, although it may be due to the extravasation of blood either in the abdominal wall or into the peritoneal cavity.

Blood Pressure. Attempts have been made to establish blood pressure criteria which could be applied in judging cases with abdominal injury, especially in respect to operability and the optimal time for operation. A systolic blood pressure of 80 has been suggested as the level below which operation is inadvisable. Lowering of the blood pressure is usually paralleled by a rise in pulse rate, but like the pulse, as an indicator of the degree or continuance of hemorrhage, blood pressure changes are undependable, because fatal hemorrhage may occur before they reflect reduction in the volume of circulating blood. Nevertheless, frequently repeated blood pressure determinations often furnish information of value in the management of abdominal casualty cases.

A method which was suggested and employed by some surgeons during and after World War I, in an attempt to "differentiate shock from hemorrhage" is described here only to be condemned as unreliable and unsafe. It consisted of making blood pressure observations following the administration of a hot infusion, which it was contended would cause a rise in blood pressure only if shock was due to hemorrhage.

CLINICAL LABORATORY STUDIES

Examination of Urine. Routine gross or microscopic examination of the urine frequently reveals injuries to the urinary tract not suspected on the basis of the projected course of the missile or from the location and nature of a blow. Absence or delay in the detection of hematuria may result from severance or obstruction of a ureter as well as suppression of urine which sometimes follows injuries to the urinary tract.

Hematologic Studies. Since changes in the composition and physical character of the blood depend not only on the rate, duration and total volume of blood loss, but are also influenced by concomitant traumatic shock due to tissue injury, and parenteral fluid therapy, blood studies following abdominal trauma often reveal only slight or confusing deviations from the normal. A degree of shock disproportionate to that which follows the external loss of a given amount of blood often occurs when the blood is spilled into the peritoneal cavity. The intraperitoneal outpouring of fluid in response to the presence of the blood, coupled with the loss of blood fluid into the tissues tend to cause hemoconcentration and this trend in the composition of the blood can mask the actual loss of red blood cells. The loss of fluids from the blood often exceeds the movement of fluids into the blood in the effort of the organism to restore and maintain blood volume. Therefore, depending on the amount, rate and duration of hemorrhage, and the degree of associated traumatic shock, as well as the fluid administered parenterally, the blood of

patients with abdominal injuries may be concentrated, diluted or practically unchanged in composition.

Red blood cell counts and hemoglobin index determinations, even though repeated after short intervals, are usually not dependable indications of either the amount or the trend of intraperitoneal bleeding, as changes in them do not occur until there has been great blood loss. Significant lowering of the red blood cell count and the hemoglobin index are ordinarily preceded by clinical evidence of continued hemorrhage.

An early and sometimes considerable increase in the blood leucocyte count may occur as a result of the presence of blood in the peritoneal cavity.

Although even repeated hematologic surveys sometimes fail to show the degree or even the trend of intraperitoneal hemorrhage, the demonstration of changes in the relative cell volume by the hematocrit method, and the estimation of the specific gravity of the blood plasma or of the whole blood either by Hammerschlag's method,[7] or by the Barbour and Hamilton falling drop apparatus,[8] usually permit rapid and early detection of the concentration changes associated with shock due to hemorrhage.[9]

Not only are mean corpuscular volume and blood specific gravity studies of value in the diagnosis and immediate therapy of patients with abdominal injuries, but these tests for dehydration and plasma protein depletion are of great assistance in the subsequent management of such cases.

Bacteriological Studies. Examination of direct smears and culture growths made from the edges of wounds or from wound exudate, furnish information which may be valuable in choosing appropriate chemotherapeutic or bio-immunologic agents in the prophylactic treatment of peritonitis and wound infection. In studying wound contamination by cultural methods, it is important that anaerobic as well as aerobic cultures be made.

X-RAY EXAMINATION

When missiles are retained within the abdomen or when there is a possibility that more than one projectile has entered either the abdomen or the abdominal wall, fluoroscopic or roentgenographic examinations are of obvious value, especially in military practice, since many of the wounds in modern warfare are caused by low velocity projectiles which are retained. The use of a portable *x*-ray apparatus is often desirable in order to avoid moving the patient, and under some circumstances, *x*-ray examination is best done in the operating room. In addition to furnishing information of value in planning the operative incision in relation to a retained missile, the *x*-ray examination may reveal gas beneath the diaphragm, the existence of a diaphragmatic hernia, or reduced diaphragmatic excursion. The absence of gas beneath the diaphragm even though a gas-containing hollow viscus has been perforated, may be due to escape of the gas through the abdominal wall wound or to the small amount of gas contained in the perforated viscus. Roentgenography of the thorax often discloses chest lesions which must be taken into account in the management of the abdominal injury. Fluroscopy ordinarily supplies sufficient information in less time than is required for roentgenography, but when more than fluoroscopic examination is necessary, anteroposterior and lateral roentgenograms usually permit satisfactory localization of foreign bodies. Stereoscopic radiograms, however, are sometimes more informative than are single projections. If precise localization is required, this can be accomplished by one of several methods devised for this purpose.

Skiagraphic observations made in a darkened operating room with the aid of a fluroscopic bonnet, are seldom necessary during operations for abdominal injuries. The method of intermittent fluoroscopic control was found to be more satisfactory in World War I for general use in the extraction of metallic foreign bodies than were electrovibrators, telephone probes, or other

similar devices, because approximately one-fifth of the foreign bodies were not magnetizable.[10]

Pyeloureterography following the intravenous injection of *x*-ray opaque solution, as well as cystography following the injection of *x*-ray opaque solution into the bladder, are helpful means of detecting not only the existence but the extent of injuries involving the kidneys, ureters and bladder.

ENDOSCOPIC EXAMINATION

Endoscopic examinations are occasionally useful when other methods fail to furnish conclusive data in patients with abdominal injuries. Proctoscopy or sigmoidoscopy may disclose an injury of the terminal portion of the large bowel, either by affording visualization of a wound which is beyond the reach of or not detectable by the examining finger, or by merely revealing the presence of blood. Esophagoscopy and gastroscopy may, under some circumstances, be employed when injury to the esophagus or the stomach is suspected. Cystoscopic examination, although not satisfactory when the bladder has been perforated, may in the presence of an intact bladder permit detection of bleeding from a ureteral orifice. Furthermore, the preoperative introduction of a large catheter into the ureter from which bleeding has been observed facilitates its repair after the abdomen has been opened.

PERITONEOSCOPY

This method of examination, which has been the subject of several reports by Hamilton,[11] has been suggested as a possible aid in diagnosis in cases with only suspected penetration of the abdominal wall. The peritoneoscope is introduced through a small incision, and the areas of the parietal peritoneum through which perforation might possibly have occurred are surveyed. If a perforation of the abdominal wall is found, exploration should be done even though no blood, gastrointestinal contents, bile, or urine is found, as it is not safe to depend upon peritoneoscopic examination to determine whether or not visceral perforation has occurred. Hamilton,[12] has recently described an apparatus which can be introduced through the peritoneoscope to permit manipulation or movement of viscera, thus increasing the range of usefulness of peritoneoscopy.

DETERMINATION OF PENETRATION BY EXPLORATION

Exploration of the Wound. When complete penetration of the abdominal wall is in doubt, it may be possible by means of a pair of blunt forceps, used as a probe, to determine whether or not the perforation extends through the parietal peritoneum. However, when a wound extends obliquely for a considerable distance in the abdominal wall, a shift in muscle, fascia, or fat may make it impossible to follow the wound throughout its entire extent, so this method of examination is not always dependable. Although the probing of wounds has been deprecated, much valuable information may sometimes safely be obtained by this simple method.

Another procedure which may be employed when there is uncertainty concerning perforation of the peritoneal membrane, consists of making an incision down to the peritoneum near the entry wound in the abdominal wall, and then, by peeling away the peritoneum in the region of the wound, the presence or absence of peritoneal perforation usually can be determined.[13]

Exploration through a Small Incision. The introduction into the rectovesical space of a sponge on a long sponge holder, through a small suprapubic incision, for the purpose of determining the presence or absence of blood, gastrointestinal contents, bile, or urine has been employed when doubt has existed concerning abdominal wall or visceral perforation. Although this method may at times demonstrate intraperitoneal injury which cannot be ascertained by other means, it is essential that no blood be allowed to enter the abdomen through the intentionally made suprapubic incision. Furthermore, this

method is not dependable when there has been extraperitoneal injury of a viscus, and even in the instance of intraperitoneal injuries may escape detection even when exploratory laparotomy is done.

Although blood, and less frequently ex-

Fig. 8. Fig. 9. Fig. 10.

Fig. 8. Hematoma located near the junction of the mesentery with the intestine. Hematomas in this location sometimes extend beneath the serous coat of the bowel and should be incised as they may obscure an intestinal perforation. The injury to terminal vasa recta responsible for the formation of hematomas in this position occasionally causes such a degree of ischemia of a small segment of adjoining intestine as to necessitate intestinal resection.

Fig. 9. Hematoma located in the central zone of the mesentery. Because of the opportunities for collateral circulation, small-sized hematomas in this region are not likely to be associated with serious impairment of the blood supply to the nearby intestine, and they should usually be left alone. However, if a hematoma in this position is of large size and especially if it can be seen to be enlarging or if there is active bleeding through an opening in it, it should be opened widely and a search made for the unarrested bleeding point.

Fig. 10. Hematoma located between the leaves of the sigmoid mesentery. Hematomas resulting from injuries to the large branches of the superior or inferior mesenteric arteries near the root of the mesentery sometimes extend over a large area in the retroperitoneal space, even reaching into the pelvis. Under such circumstances, location of the bleeding point is difficult or impossible, and because of the danger of exposing the retroperitoneal space to infection and the possibility of causing more blood vessel injury, search for the torn vessel should usually be done only when it is believed or known that a damaged vessel is still bleeding.

injuries of the large intestine, there is often so little hemorrhage, and the intestinal contents may be of such solid consistency and small amount that gravitation of blood or feces into the rectovesical space may be long delayed or even may never occur.

Exploratory Laparotomy through a Large Incision. When doubt exists despite the employment of other methods for determining whether or not there has been peritoneal perforation or visceral injury, early exploration is usually safer than prolonged observation and conservative treatment. Even though a gas-filled hollow viscus has been perforated there is usually an absence of the hissing sound often heard when the peritoneum is opened following rupture of a peptic ulcer, the gas having already escaped through the abdominal wall wound. Because of an obscuring hematoma (Figs. 8, 9 and 10), inaccessibility, or the haste with which operation is done, visceral travasated gastrointestinal contents, may be found as soon as the peritoneum is incised, the location of the injury may be so distant, or the amount of free fluid in the part of the abdominal cavity first brought to view may be so small that until the incision has been enlarged and exploration has been carried farther, it is not possible to determine whether there has been visceral injury. Partial mobilization of the duodenum or of fixed segments of the large intestine, and examination of the posterior surfaces of the stomach and the transverse colon after opening the lesser peritoneal cavity, may be required to expose retroperitoneal or other hidden injuries. (Figs. 11, 12, 13.)

WOUNDS OF SPECIFIC ORGANS OR STRUCTURES

Several generalities concerning the likelihood of injuries to various organs are well

known. Injuries caused by blunt force, especially when directed against the upper portion of the abdomen or the lower chest, in respect to fixation of the several portions of the large intestine, as well as the proximity of some parts of the colon to the bony

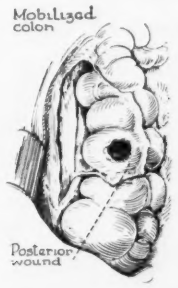


Fig. 11.

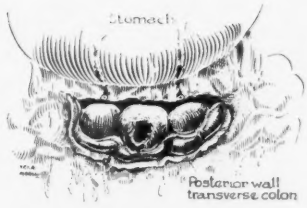


Fig. 12.

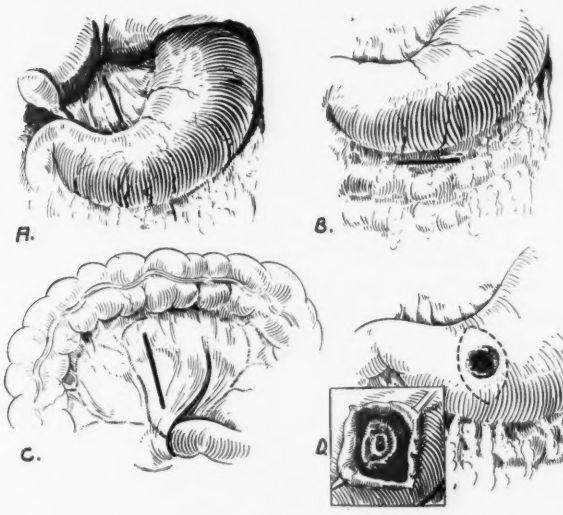


Fig. 13.

Fig. 11. Partial mobilization and reflection of the ascending colon to reveal a retroperitoneal perforation.

Fig. 12. Partial mobilization and rotation of the transverse colon to reveal a perforation on its posterior aspect.

Fig. 13. Various methods of exposing a wound of the posterior wall of the stomach. Shown in (A), (B) and (C) are incisions for opening the lesser peritoneal cavity. Exposure of a posterior wall injury by an opening in the anterior stomach is shown in (D).

often involve the liver, the spleen, the kidneys or the diaphragms, which structures, because of their turgor, size, fixation, and the presence of overlying ribs, are particularly vulnerable. The position and fixation of the duodenum and the pancreas make these structures susceptible to injury by anteriorly directed blunt forces. The stomach, by virtue of its size and comparative immobility, is likely to be injured either by projectiles or by blunt force. The small intestine, although relatively mobile, is, by its very extent, subject to injury, especially perforating wounds. Variations pelvis, influence the frequency of injuries to this segment of the alimentary tract.

Wounds of the Abdominal Wall. Wounds in which a missile or sharp object pierce the abdominal wall without causing either hollow or solid visceral injury may result fatally either because of hemorrhage or peritonitis. Signs and symptoms which are either suggestive or definitely indicative of peritoneal irritation are likely to exist as a result of the presence of blood or of the occurrence of peritonitis, and it is often only by direct exploration that the absence of visceral injury can be determined.

Esophagus. Wounds of the lower end of the esophagus are usually associated with thoracic injuries. Persistent nausea

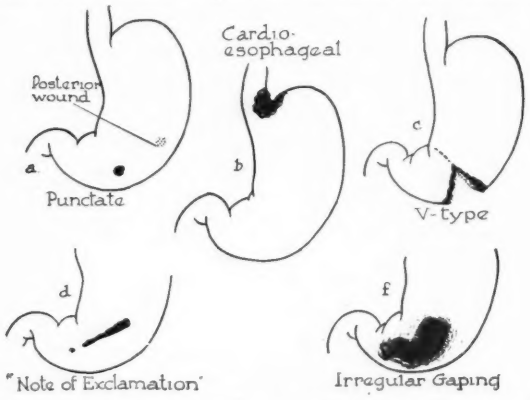


FIG. 14. Diagrammatic representation of some of the more common types of stomach wounds.

and vomiting may accompany a lesion of the esophagus, but there are no characteristic signs or symptoms to indicate esophageal injury, and recognition usually depends on projecting the course of the missile or instrument causing injury, or upon exploration. Damage to the esophagus may be revealed by esophagoscopic examination, but this method of diagnosis is ordinarily not feasible or satisfactory.

Stomach. Stomach wounds comprised about 7 per cent of all abdominal injuries coming to the hospitals during the 1914 to 1918 World War.[10] Two-thirds of the gastric lesions were unaccompanied by other visceral injury discoverable at operation. Other organs most frequently injured were: the small gut, liver, colon, kidney and the spleen, in the order named. Some of the more common types of stomach wounds are shown in Figure 14.

The wounds are usually two in number, and are most frequently situated on the anterior and posterior walls. If the anterior opening is small, and if the organ was not distended at the time of injury, there may be no protrusion of the mucous membrane and no escape of gastric contents. Leakage may occur into the lesser sac, even when none is present anteriorly. If but a single wound is present, it is usually located on the anterior wall.

There are no distinctive signs or symptoms of gastric injury. Pain may be intense if the stomach was filled at the time of injury and if a considerable amount of acid

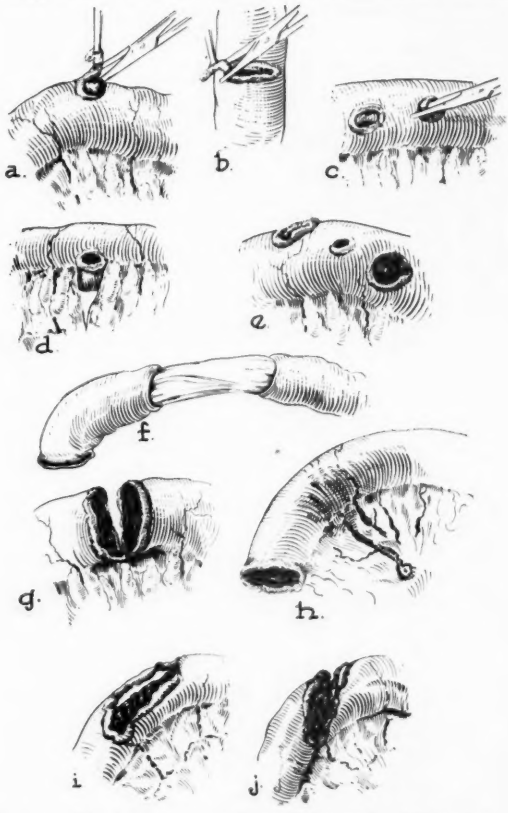


FIG. 15. Various types of small intestinal injuries. (*a*) Single punctate wound which may be closed by purse-string suture after removing everted mucosa. (*b*) Single transverse lacerated wound. (*c*) Closely situated punctate wounds which may be communicated and then closed as a single wound. (*d*) Wound at junction of mesentery with intestine. Such wounds may be obscured by a mesenteric hematoma. (*e*) Multiple punctate and lacerated wounds. In most instances, these should be closed individually. Resection of the bowel for such injuries should be done only when separate closure of the perforations is not feasible. (*f*) Injury in which mesentery has been torn away and muscular coats stripped back. (*g*) Practically complete division of the bowel. This type of injury may be produced even when a comparatively small missile strikes a collapsed loop. (*h*) Wound of the mesentery with thrombosis of vasa recta and infarction of bowel wall. (*i* and *j*) Extensive lacerated wounds of the sort which sometimes necessitate resection of a small segment of intestine. The extent and character of hollow viscera wounds caused by projectiles is determined not only by the size and shape of the missile, but by the distention or collapsed state of the viscus at the time of injury.

stomach contents has spilled into the peritoneal cavity. Severe vomiting and

nausea may accompany gastric wounds, but these symptoms may be absent, and hematemesis is unusual following stomach injuries. When there has been extravasation of stomach contents, tenderness and rigidity can usually be detected. If a sufficient amount of gas from the stomach wound is retained in the peritoneal cavity, percussion reveals obliteration or reduction of the normal areas of liver dullness. It may also be possible to demonstrate the free gas in the peritoneal cavity by *x*-ray examination.

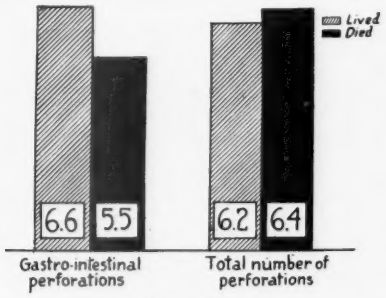


Fig. 16.

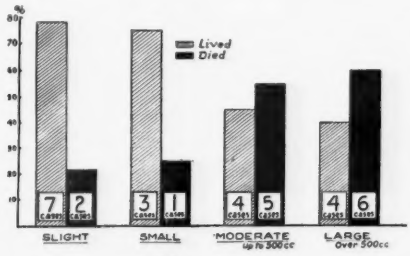


Fig. 17.

Fig. 16. Graphic representation of the average number of visceral perforations in relationship to the mortality in a series of forty-six penetrating bullet and stab wounds of the abdomen. Comparison of this Figure with Figure 17 reveals that the mortality is more directly related to the degree of hemorrhage than to the number of perforations.

Fig. 17. Graphic representation of the direct relationship between the amount of hemorrhage and the mortality in a series of cases with penetrating bullet and stab wounds of the abdomen. Comparison of this Figure with Figure 16, which latter shows the average number of perforations in relationship to mortality, emphasizes the importance of hemorrhage rather than the number of perforations in determining the outcome in penetrating abdominal wounds.

Small Intestine. The proportion of small intestinal wounds, both complicated and uncomplicated to all abdominal lesions in World War I was approximately 22 per cent.[10] Associated injuries to other viscera in the order of their frequency are those of the colon, the stomach, the urinary bladder, the liver, the kidneys, the rectum and the spleen. Duodenal injuries are infrequent, constituting approximately 6 per cent of all small gut wounds. Injuries to the jejunum comprise about 23 per cent and those to the ileum approximately 71 per cent. The number of injuries produced by a single projectile may vary from one to thirty or more, but usually there are four to six. Figure 15 shows several types of small intestinal wounds.

No characteristic symptoms are associated with injuries of the small intestine; the symptoms vary with the amount of hemorrhage, the amount of spillage of intestinal contents, co-existing wounds of other viscera, and the time elapsed since injury. Neither the size nor the number of intestinal wounds bear a constant relationship to the signs or symptoms which accompany them. Figure 16 shows the absence of any significant relationship between mortality and the number of intestinal perforations in the author's series of cases. The importance of hemorrhage rather than the number of perforations in relation to mortality is shown by comparing Figure 16 with Figure 17.

Wounds of the Colon. Wounds of the colon constituted about 22 per cent of all intra-abdominal visceral injuries in the war of 1914 to 1918.[10] The colon is not uncommonly the only part of the alimentary tract hit. The comparatively small number of reported splenic flexure wounds is remarkable and it has been suggested that because of the difficulty of differentiating between the transverse and descending colons and the splenic flexure, some of the

injuries of the two former parts should have been described as lesions of the latter.

If a colon wound is small, there may be practically no early symptoms, whether the injury is extraperitoneal or intraperitoneal. This is especially true if the latter type of wound is uncomplicated and of such small size that there has been little bleeding or spillage of the usually semisolid fecal material contained in the large intestine. It has been observed that the appearance of patients with large wounds of the colon often simulates that caused by severe hemorrhage.

Wounds of the Rectum. Injuries of the rectum are comparatively infrequent and constituted only 2.4 per cent of the lesions of abdominal viscera in World War I.[10] Associated lesions which may be encountered are those of the urinary bladder and the pelvic colon, or less frequently, injuries of the small bowel.

The rectal lesions vary in size from small perforations caused by minute projectiles or fragments of bone, to extensive lacerations. It is sometimes possible by digital examination to detect a perforation or a spicule of bone which projects into the lumen, while in other instances, blood on the gloved examining finger may be the only indication of rectal injury. Proctoscopic examination may reveal a perforation which is not detectable by an examining finger; and even if a perforation is not seen, the presence of blood as revealed by proctoscopic examination is indicative of injury to the rectum.

Wounds of the rectum are likely to be accompanied by fractures of the bony pelvis, spicules of which may wound the gut. As in the instance of the rest of the large intestine, there may be remarkably few symptoms associated with small wounds of the rectum. When a projectile or other object responsible for rectal trauma has entered through the hip, perineal, sacral, or gluteal regions, the existence of a rectal wound may be overlooked and it is sometimes not until there are evidences of general sepsis, pelvic cellulitis, and even localized or diffuse peritonitis, that it is realized that the rectum has been injured.

Injuries of the Gallbladder and Bile Ducts. On account of their small size, injuries to these structures are relatively infrequent. Although spillage of bile following perforation or rupture of the gallbladder may result in considerable peritoneal irritation, as a rule the symptoms produced by these injuries are not characteristic, and it is only through a consideration of the probable wound track or by finding at operation that injuries of these structures are suspected or discovered. Bradycardia due to the outpouring of bile into the peritoneal cavity has been reported, but most patients with such injuries have a rapid pulse due to shock, and death usually occurs before there is slowing of the pulse.

Injuries of the Mesentery and Omentum. Injuries of the great omentum and the mesenteries are common. No characteristic symptoms are produced by injuries to these structures but hemorrhage due to injury of the vessels which course in them is often responsible for much of the shock which exists in patients with traumatic lesions of the abdomen, and frequently much of the blood found in the peritoneal cavity at the time of operation has come from injured vessels in the omentum or the various mesenteries.

Wounds of the Liver. Because of its large size, the liver is often injured, and wounds of this organ composed 13.3 per cent of all traumatic abdominal lesions in World War I.[10] Approximately three-quarters of the liver injuries were uncomplicated. Associated wounds to be expected are, in the order of their frequency, wounds of the colon, stomach and kidney. Figure 18 shows various types of liver injuries. The possibility of injury to the liver must be considered in all abdominothoracic wounds on the right side of the body. In addition to liver injuries caused by penetrating projectiles, serious live trauma frequently results from fractured ribs and from blunt

force. "Blast" injuries of the liver have also been reported.

Hemorrhage is always present following if the patient survives the immediate loss of blood.

Wounds of the Spleen. Wounds of the

Fig. 18. Fig. 19. Fig. 20.

Fig. 18. Illustrations of various types of wounds of the liver. Excepting punctate wounds, indicated in (*b*), and furrowing or gutter wounds, indicated in (*d*), these different sorts of liver damage may be caused by blunt force as well as by projectiles or indriven objects.

Fig. 19. Illustrations of several types of injuries of the spleen. Excepting the type of wound shown in (*d*), such lesions may be produced by blunt force as well as by projectiles or other indriven objects. Splenectomy is the only satisfactory procedure when lesions such as those shown in (*a*) and (*b*) are found, and because of the danger of secondary hemorrhage, removal of the spleen should usually be done when a subcapsular hematoma such as is indicated in (*c*) is present, even though there is no bleeding from the spleen at the time of operation. When a small punctate wound is located near one of the poles of the spleen, as in (*d*), and there is no bleeding from the wound at the time of operation, it may be best not to perform splenectomy, especially if there are many injuries to other organs or if the patient is suffering from shock and hemorrhage.

Fig. 20. Illustration of various types of kidney injuries. For the smaller wounds, entirely conservative methods or suture yield the best results. Even when the extensive character of a kidney wound requires nephrectomy, it is often advisable to delay this procedure for several days or at least until the patient has recovered from shock.

liver wounds, and may vary from a slight ooze to a severe and rapidly fatal loss of blood. Symptoms and signs of peritoneal irritation result from the presence of blood, and sometimes from bile extravasated into the peritoneal cavity. If the amount of bleeding has been considerable, dullness may be detected in the flank, particularly on the right side. In the instance of cases seen two or three days after the injury, there is frequently slight jaundice; late jaundice indicates sepsis. Secondary hemorrhage from the liver may occur several days following injury. The occurrence of such delayed hemorrhage is commonly accompanied by pain, distention of the abdomen, rise of temperature, and acceleration of the pulse, associated with pallor, restlessness, and a rapid loss of strength. With these general symptoms, a localized swelling which may be indistinguishable from a secondary abscess usually develops

spleen are much less frequent than those of the liver, only forty-nine having been recorded as occurring in the American Expeditionary Forces in World War I.[10] Many of the cases with splenic wounds had complicating lesions, which usually involved the kidneys, stomach and colon. Figure 19 illustrates several types of injuries of the spleen.

Splenic injury must always be considered when there has been severe trauma on the left side of the chest or abdomen, whether due to blunt force or to a penetrating wound; and the possibility of perforation of the spleen by a fractured rib must be kept in mind.

The manifestations of injuries of the spleen are principally those of shock and hemorrhage, along with signs of peritoneal irritation caused by blood in the peritoneal cavity. An accumulation of blood in the left flank may occur in nonpenetrating

wounds of the abdomen with splenic injury, but damming up of blood in the left loin is not frequently detectable.

Wounds of the Pancreas. Cases with wounds of the pancreas reaching the hospital comprised about 0.2 per cent of all abdominal injuries in World War I.[10] In addition to wounds caused by projectiles, injury of the pancreas may be due to impingement of that organ against the vertebral column as the result of blunt pressure, such as that caused by being thrown against a motor vehicle steering wheel, or by being rolled over by a vehicle.

Pancreatic injuries do not cause characteristic early signs or symptoms. Even at the time of operation, the lesions are often obscured by a retroperitoneal hematoma. The first evidence of a wound of the pancreas may be that produced by the development of a pseudocyst several weeks or longer after the injury. Elevated levels of blood sugar, as well as increased amounts of blood lipase and diastase may be present for several days following pancreatic trauma.

Wounds of the Adrenal Glands. Due to their small size, the adrenal glands are seldom wounded. Even when damage to these glands occurs, recognition of the injuries is difficult or impossible because of the lack of distinctive symptoms, the position and smallness of the glands, and the frequent presence of an obscuring hematoma.

Injuries of Large Blood Vessels. Injuries to large abdominal blood vessels usually result in death before patients with such wounds can be transported to a hospital or to a military surgical unit. In addition to the immediate signs of severe shock and hemorrhage, as well as peritoneal irritation caused by hemorrhage into the peritoneal cavity, there may also be bleeding into the retroperitoneal spaces; and when this occurs, ileus is likely to be pronounced.

Abdominothoracic Wounds. These complicated wounds comprised 4.6 per cent of all the thoracic injuries brought to the evacuation hospitals of the American Expeditionary Forces in World War I.[10] Other statistics,[6] based on observations in that war, show that the percentage of wounds involving both the chest and the abdomen was about 12 per cent. Abdominothoracic wounds are most often confined to one side, but both sides of the chest may be involved. Wounds low enough to involve both diaphragms are usually immediately fatal. In approximately one-third of the cases, a hollow abdominal organ is penetrated, and liver wounds are more common than are those of the spleen.

The characteristic manifestations of abdominothoracic wounds are sudden pain in the abdomen, dyspnea, hemothorax, abdominal rigidity and shock, which are dependent upon both hemorrhage and interference with normal respiration. Vomiting may occur, and there may also be localized tenderness. In cases with relatively small chest wounds, tension pneumothorax may cause more serious respiratory and circulatory disturbance than is ordinarily produced even by "blowing" wounds.

Wounds of the Kidneys. The kidneys may be injured either by blunt force or by penetrating missiles. These injuries constituted 6.3 per cent of all abdominal injuries in World War I, and one-half of the cases were uncomplicated.[10] Various types of kidney injuries are shown in Figure 20.

Hematuria is present in about 90 per cent of cases. It is usually detectable immediately after the injury and is, as a rule, proportionate to the extent of renal damage. Routine examination of the urine in penetrating or crushing wounds of the abdomen often reveals unsuspected urinary tract injuries. Although there may be grossly detectable blood in the urine, microscopic examination of the sediment from a centrifuged specimen may be necessary to reveal red blood cells. Hematuria may not be detectable for quite some time following kidney injury, either because of urinary suppression, the presence of blood clots or pieces of kidney tissue in the ureter, or severence of the ureter. Although hematuria may persist for a

relatively long time, it usually continues for only a few days.

Except when there is much associated hemorrhage, kidney injuries are attended with remarkably little shock. Pain is a common symptom and is often of a colicky nature, due to the presence of blood clots in the ureter. If there is distention of the pelvis or the true capsule of the kidney, however, pain is likely to be of a dull character. The pain may be generalized over the abdomen or it may radiate to the lumbar or sacral regions, or to the shoulder as well as to the hip. Nausea and vomiting are frequent symptoms, even when there has been no extravasation of blood or urine into the peritoneal cavity. Hemorrhage may occur from the loin wound or a hematoma may form in the flank. There may be localized rigidity of the lumbar muscles, or there may be considerable bleeding into the peritoneal cavity, so that tenderness, rigidity and dullness due to free blood in the abdomen may be detected.

Secondary hemorrhage is a fairly frequent and sometimes fatal late complication. It occurs most often during the second and third weeks after injury, usually in cases in which the urine is contaminated. Delayed hemorrhage may start afresh, or may manifest itself as an exacerbation of a persistent primary hemorrhage. Unlike primary hemorrhage, recurrent bleeding is often accompanied by clotting of the blood in the bladder.

Extravasation of urine into the peritoneal cavity, by adding to peritoneal irritation, increases the degree of rigidity and tenderness. External leakage of urine may occur when the pelvis of the kidney has been opened or when the ureter has been torn, but seldom is present when the injury involves the renal parenchyma alone. Especially in late cases, the escape of urine through a loin wound or the detection of a uriniferous odor furnishes evidence of a kidney injury.

X-ray examination is valuable in revealing the absence of a psoas muscle outline, flexion of the spine towards the affected side, change in the size and contour of the kidney outline and limitation of renal mobility. Intravenous or retrograde pyelography or pyeloureterography may demonstrate the character and extent of a renal injury. Anterior displacement of the ureter

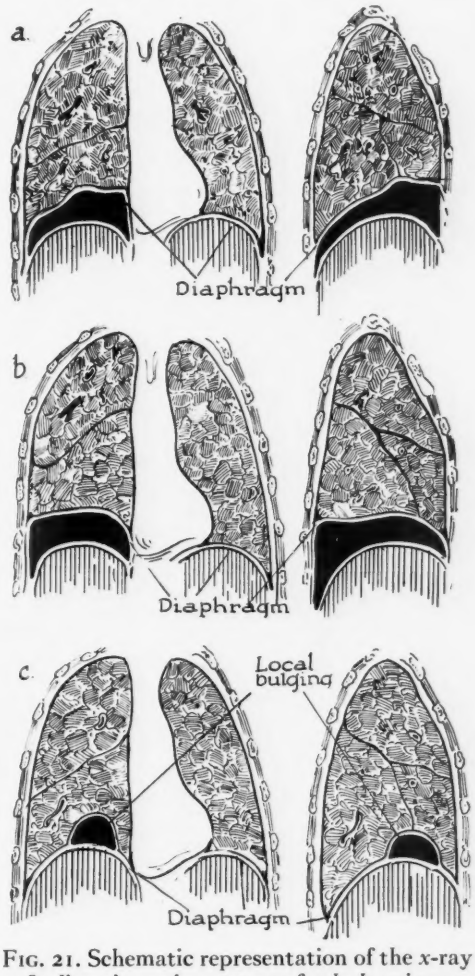


Fig. 21. Schematic representation of the *x*-ray findings in various types of subphrenic suppuration, and in abscess of the liver. (*a*) Shows the typical findings when subdiaphragmatic suppuration is secondary to liver abscess. There is obliteration of the cardiophrenic angle in the P. A. view and of the anterior costophrenic angle in the lateral view. (*b*) Shows the typical findings when subdiaphragmatic suppuration is due to peritonitis from various causes, including abscess of the liver. There is obliteration of the costophrenic (instead of the cardiophrenic) angle in the P. A. view and the posterior costophrenic (instead of the anterior costophrenic) angle in the lateral view. (*c*) Shows the typical findings in a liver abscess which has not ruptured into the subphrenic space.

revealed by a lateral pyelogram, is evidence of a perinephritic accumulation of pus, blood or urine.

Wounds of the Ureters. Wounds of the ureters are rare, but preoperative recognition of their existence is important, for the introduction of a ureteral catheter usually facilitates location and repair of the injured ureter after the abdomen has been opened. Determination of the presence of a ureteral wound is often difficult. The appearance of hematuria may be delayed by spasm of the ureter, suppression of urine, severence of the ureter, obstruction by blood clots or by the existence of an anomaly.

Wounds of the Bladder. Wounds of the bladder comprised approximately 5 per cent of all abdominal lesions in World War I and one-half of the cases were uncomplicated.[10] Rupture of the bladder with extensive laceration due to a "bursting effect" is likely to occur if it is distended at the time of injury by a blunt force, but this same type of injury may also be produced when the full bladder is struck by a small projectile. The majority of bladder wounds were complicated by intestinal or bone injuries or both. A wound of the rectum was found in from 10 to 15 per cent of the cases, but injury of the prostate was comparatively rare. The small intestine was the organ most frequently wounded in conjunction with the bladder. In cases reaching the hospital, the entrance wound was most frequently located posteriorly, and a wound of exit usually was not present.

There may be hematuria following either penetrating or nonpenetrating bladder wounds, but micturition is sometimes impossible. Vomiting is a frequent symptom in extraperitoneal wounds. Discharge of urine through the entry wound is rather uncommon.

Bleeding into the bladder suggests an extraperitoneal lesion and with this type of injury there may be obvious swelling above Poupart's ligament, and at operation a large hematoma is sometimes found in the vicinity of the wound in the viscus. An empty bladder, especially when associated with signs of peritoneal irritation, suggests the existence of intraperitoneal perforation with leakage of urine into the peritoneal cavity.

Cystoscopic examination is frequently impossible if there has been intraperitoneal perforation of the bladder. A cystogram may reveal the existence and extent of bladder wounds, especially the extraperitoneal ones associated with fractures of the bony pelvis. Extraperitoneal bladder lesions are often followed by pelvic cellulitis. In long-standing cases, bone necrosis, calculus formation and cystitis are frequent complications.

SELECTION OF CASES FOR OPERATION

The following criteria may be generally applied in determining the need for or the advisability of operative treatment in cases which have sustained abdominal trauma.

Cases Definitely Requiring Operation. When there is known or suspected abdominal visceral injury of recent occurrence without associated severe shock or extensive injuries to other parts, celiotomy should be performed as soon as possible.

Hopeless Cases. When the abdominal injury is of long duration, or when it is so extensive that satisfactory anatomical repair or functional restitution is obviously impossible, operation is not only of no avail but, in military practice, consumes time and materials which could be employed in caring for other casualty cases.

Cases Not Requiring Operation. In contrast to the cases in which the time factor or the extent of the injury make operation seem hopeless are those in which even exploration is unnecessary. Elective nonoperative management is most often applicable when no hollow visceral injuries are considered to exist, as when the injury is limited to the upper abdomen, especially to the liver area, and particularly if the injury has been inflicted by a small projectile or instrument.

When elective nonoperative treatment is contemplated, it is important to attempt

determination of whether or not hemorrhage has ceased, employing the methods already described. As shown by the observations of Kekwick, Marriott, Maycock, and Whitby,[14] if there are evidences of shock, differentiation must be made between primary shock from which improvement, including return of blood pressure to near normal, rapidly occurs following simple measures such as rest, the application of external heat, and the administration of morphine, and in which transfusion of even plasma may not be necessary; and secondary shock due to continued hemorrhage, in which improvement does not follow simple supportive treatment, and in which large transfusions of plasma or whole blood are required.

Borderline Cases. When twelve or more hours have elapsed since the injury, operative intervention usually only hastens death, regardless of the extent of the abdominal lesion. When wounds involve only the upper abdomen, however, operation even after a considerable lapse of time is sometimes advisable, not only because of continued hemorrhage from the liver or spleen, but because the frequent absence of hollow visceral perforations makes late operation relatively safe. Although injuries caused by small missiles may not require exploratory celiotomy, apparently trivial external lesions are often accompanied by extensive intraperitoneal wounds or gaping lacerated wounds of the hollow viscera.

CRITERIA FOR INTESTINAL RESECTION

Intestinal resection is to be avoided whenever possible, but several more or less absolute indications for removing a segment of the bowel must be recognized. Resection of a part of the intestine is most frequently necessary because of interference with its blood supply resulting from detachment of the mesentery, or because of division or thrombosis of large mesenteric blood vessels. If not at once evident, these conditions are usually manifested by absence of pulsation in mesenteric arteries or by changes in the adjacent bowel, including deviation from normal color and absence of motility even after squeezing or warm applications. Resection may also be required when there is devitalization due to severe contusion or crushing of the intestine, or when there are multiple wounds in such close proximity that their individual closure would result in reduction of the size of the lumen or angulation of the bowel sufficient to cause obstruction.

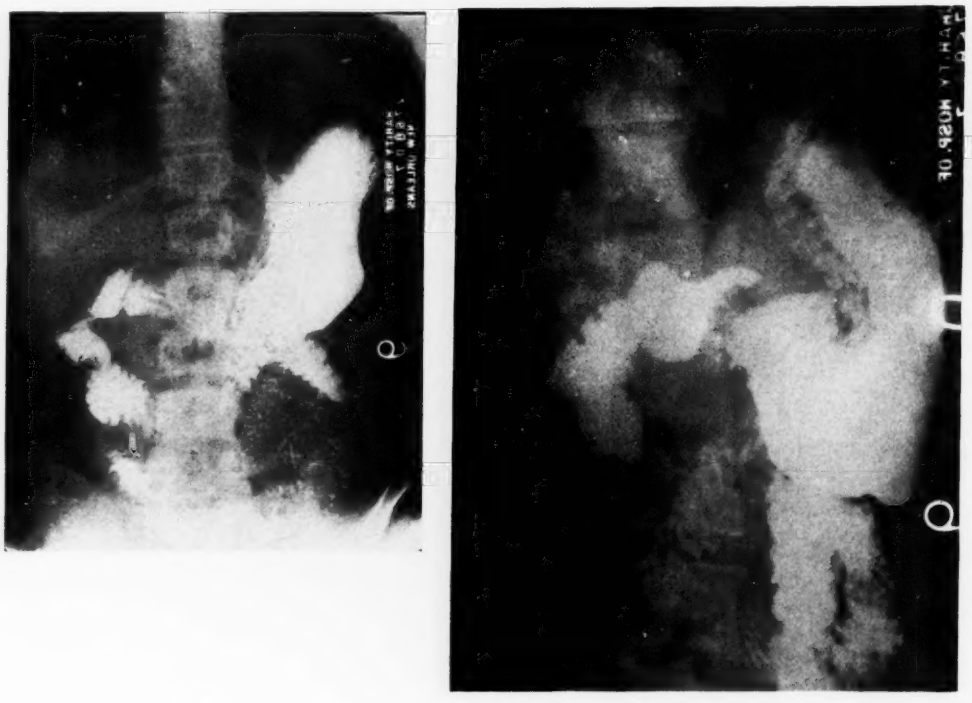
POSTOPERATIVE OR LATE COMPLICATIONS

In addition to early complicating conditions, such as shock and hemorrhage, which occur simultaneously with or immediately following injury, late complications must be anticipated and recognized promptly.

Shock and Hemorrhage. Incomplete recovery from an initial state of shock, delayed shock, or recurrent shock following operation, is frequently observed following traumatic lesions of the abdomen. Secondary shock may be due to continuance of bleeding from kidney, liver or other wounds, or may result from incomplete hemostasis at the time of operation. Massive secondary hemorrhage and severe shock may, in other instances, be due to the rupture of a subcapsular hematoma, particularly of the spleen or liver, after several days or even weeks have elapsed following the injury. Details of the evidences of secondary bleeding from the liver have already been discussed in conjunction with the special consideration of liver injuries.

Ileus and Peritonitis. Some degree of both adynamic ileus and peritonitis follows many abdominal traumas, either as a result of exposure, manipulation, or contamination of the peritoneum and the peritoneal contents. Ileus may be due to the presence of a retroperitoneal hematoma, while in other instances, both ileus and peritoneal irritation may be caused by the presence of blood, bile, or urine in the peritoneal cavity. Mechanical intestinal obstruction may complicate cases of abdominal trauma in which there has been associated peritonitis, or when operative

repair of gastrointestinal injuries has resulted in either narrowing of the lumen or angulation of the intestine. Discontinuance of the practice of performing enterostomy and of introducing drains into the peritoneal cavity, has eliminated these causes of secondary intestinal obstruction.



A B

FIGS. 22A AND B. A traumatic pseudocyst of the pancreas is outlined by the displacements and filling defects of the stomach and transverse colon in these anteroposterior and oblique *x*-ray projections following ingestion of barium emulsion by a patient whose pancreatic injury was incurred in an automobile accident.

Thoracic Complications. Pulmonary complications, notably lobular or massive pulmonary atalectasis and pneumonitis, must be anticipated and recognized early by means of physical or roentgenologic examination.

Dehydration. Dehydration or insufficient fluid intake can be detected by measurement of the urinary output, dryness and coating of the tongue, or by such laboratory methods as red blood cell counts, hematocrit determinations, or blood specific gravity studies.

Hypoproteinemia. Lowering of the plasma proteins to levels where wound healing and convalescence in general is retarded can be quickly and simply recognized by means of the falling drop method for determining blood specific gravity, combined with determinations of mean corpuscular volume by the hematocrit.

Wound Infection. Recognition of wound infections due to common pyogenic organisms, gas bacilli and other anaerobic or micro-aerophilic bacteria including the micro-aerophilic hemolytic streptococcus, can often be made with fair accuracy by inspection or palpation of the wound, by the appearance of the exudate, or by the associated odor, which latter may be extremely foul when anaerobic infection exists. Retroperitoneal infection may give few evidences of its existence other than profound toxemia. Bacteriologic studies including examination of cultures and direct smears made from the wound edges or wound exudate can furnish additional data which permit employment of the most appropriate therapeutic agents, such as specific sera or vaccines, sulfonamide drugs, oxidizing substances such as zinc peroxide, or such chemotherapeutic preparations as gramicidin and penicillin.

Residual Abscess. Continued elevation of temperature or the occurrence of chills, when not accounted for by wound infection, suggests the presence of a residual abscess. An abscess in the rectovesical space may also be accompanied by diarrhea, loss of tone of the anal sphincter, and a pelvic mass detectable by rectal examination. Subphrenic abscess formation may be revealed by the presence of tenderness when pressure is made over the lower ribs, and the existence of an abscess in this region may be confirmed by the roentgenologic findings described by Granger[15] and shown in Figure 21. Subhepatic, perirenal, iliac fossa and other residual intraperitoneal abscesses are usually detectable by the presence of localized tenderness and rigidity, or even a palpable mass. *X*-ray examination may reveal a retained foreign body.

Rupture of Intramural Abscess and Delayed Rupture of the Intestine. Rupture of an intramural abscess may occur into the peritoneal cavity, and delayed rupture of the intestine may follow the stripping loose of its outer layers at the time of injury, as well as infarction resulting from tearing away or other injury of blood vessels in the mesentery. The former complication rarely occurs before the sixth or seventh day following injury or operation and usually causes immediate severe prostration and profound toxemia which is ordinarily soon followed by death. Delayed rupture of the intestine, although usually more insidious in its manifestations as a result of walling off by adjacent viscera, is no less fatal than is intraperitoneal rupture of an abdominal wall abscess. Ischemic necrosis and rupture of the intestine may occur as early as the third day following injury or operation, but is ordinarily delayed for six or seven days, when it may be the first definite evidence that severe visceral damage was done at the time of an abdominal injury.

Persistent Draining Sinuses. The development and persistence of a sinus tract suggests the presence of a retained missile or other foreign body, for which *x*-ray examination or even direct exploration may be necessary.

Traumatic Pseudocyst of the Pancreas. This late complication of abdominal trauma may be expected following severe blunt injuries to the upper abdomen, or definitely known pancreatic penetration, and is detectable by palpation of a mass in the upper abdomen, or by the roentgenologic demonstration of displacement of the stomach and transverse colon. (Fig. 22.)

Toxic Manifestations of Sulfonamide Drugs. The frequent necessity of administering the various sulfonamide drugs has added the toxic effects of these agents to the possible late complications in traumatic lesions of the abdomen. Hyperpyrexia, nausea, vomiting, diarrhea, jaundice and dermatitis are common clinical evidences of toxicity due to sulfonamide therapy. Undesirably high blood concentrations of these drugs will be revealed by the laboratory studies which should govern their administration.

SUMMARY

1. Diagnosis in traumatic lesions of the abdomen includes investigation in respect to shock, hemorrhage and other associated conditions, as well as determinations of the location and extent of abdominal injury.

2. Even in obscure cases with either penetrating wounds, or visceral injuries caused by blunt force, diagnosis is usually possible before shock and hemorrhage, or extravasation of gastrointestinal contents have been fatally prolonged or extensive.

3. The clinical manifestations as well as the findings obtained by clinical laboratory, *x*-ray and endoscopic methods of study, which permit early diagnosis and serve as a guide to therapy are presented.

4. The indications for and methods of exploring the abdominal wall wound as well as the peritoneal cavity are detailed.

5. Criteria for the selection of cases requiring operative treatment, and for the timing of surgical intervention are given.

6. The incidence and character of lesions as revealed at the time of operation is reviewed.

7. Methods of determining the presence and extent of hidden lesions at the time of operation are discussed.

8. The complications which frequently follow abdominal injuries are considered in respect to the importance of their early recognition.

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TRAUMATIC AND INFECTIOUS TENOSYNOVITIS

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A SYSTEMIC disease that causes the hospitalization of 7 to 111 per hundred thousand admissions, depending on the type of hospital one investigates,[1] would hardly seem worthy of extensive discussion. Yet because the larger number are not hospitalized and because of the severe disability that flows from these affections, and because of the large percentage of errors in diagnosis, we believe that it is worthy of a careful review. Seven facts of tendon anatomy should be remembered:

First: Most tendons do not have a sheath but operate through primitive areolar tissue.[2] Here arises most mistakes in diagnosis. One may have tenosynovitis of only certain tendons.

Second: Wherever tendons began to operate under gravitational pressure beneath retentive ligaments mucous sheaths were developed[3] to (a) facilitate motion, (b) reduce wear, and (c) promote repair. This was true in the weightbearing shoulder, the hands and the feet. Specifically, tendon sheaths occur around the following tendons:[4] (Fig. 1.)

1. Shoulder—the long head of the biceps
2. Hand and wrist
   - (a) Flexor surface
     - 1′. Communal sheath for the carpal portion of the deep and superficial finger flexors 2–4, and the entire tendon of the fifth finger
     - 2′. The long flexor of the thumb
     - 3′. Digital sheaths for the tendons of fingers 2–4
   - (b) Extensor surface
     - 1′. Carpal sheath for each wrist extensor
     - 2′. Communal sheath for the carpal portion of the finger extensors 2–4
     - 3′. Extensor of the fifth finger
     - 4′. Long extensor of the thumb
     - 5′. Communal sheath for the long abductor and short extensor of the thumb
3. Foot
   - (a) Dorsal surface
     - 1′. Communal sheath for the extensors of the toes 2–5, and the peroneus tertius
     - 2′. Extensors of the great toe
     - 3′. Anterior tibial
   - (b) Mesial surface
     - 1′. Posterior tibial
     - 2′. Long toe flexors
     - 3′. Long flexor of the great toe
   - (c) Plantar surface
     - 1′. Sheath for the digital portion of each toe's flexors
     - 2′. Plantar portion of the peroneus longus
   - (d) Lateral surface
     - 1′. Communal sheath for the peroneus longus and brevis

In man the shoulder and wrist have long since ceased to be under major gravitational strain but the primitive mechanism remains. One, therefore, cannot properly speak of tenosynovitis of the tendo-achilles or of the hamstrings or of the supraspinatus.

Third: It is generally believed[2] that at first simple bursa developed at the site of friction, and as the force continued bony grooves developed and the tendon became almost completely surrounded by the bursa leaving a mesotendon between the visceral and parietal portions of the bursa on the side with minimal pressure. Nerves and vessels passed through the mesotendon. The embryological work of Shield,[3] however, shows that the synovial covering of the tendon is a "specific functional characteristic of the mesenchymal cells in-

volved and is not merely the result of a collection of fluids in the tissue." He has also shown that the development begins in the metatarsal area in the foot and extends proximally, but that even in large embryos the proximal end of the sheath is not completely walled off from the tissue spaces.

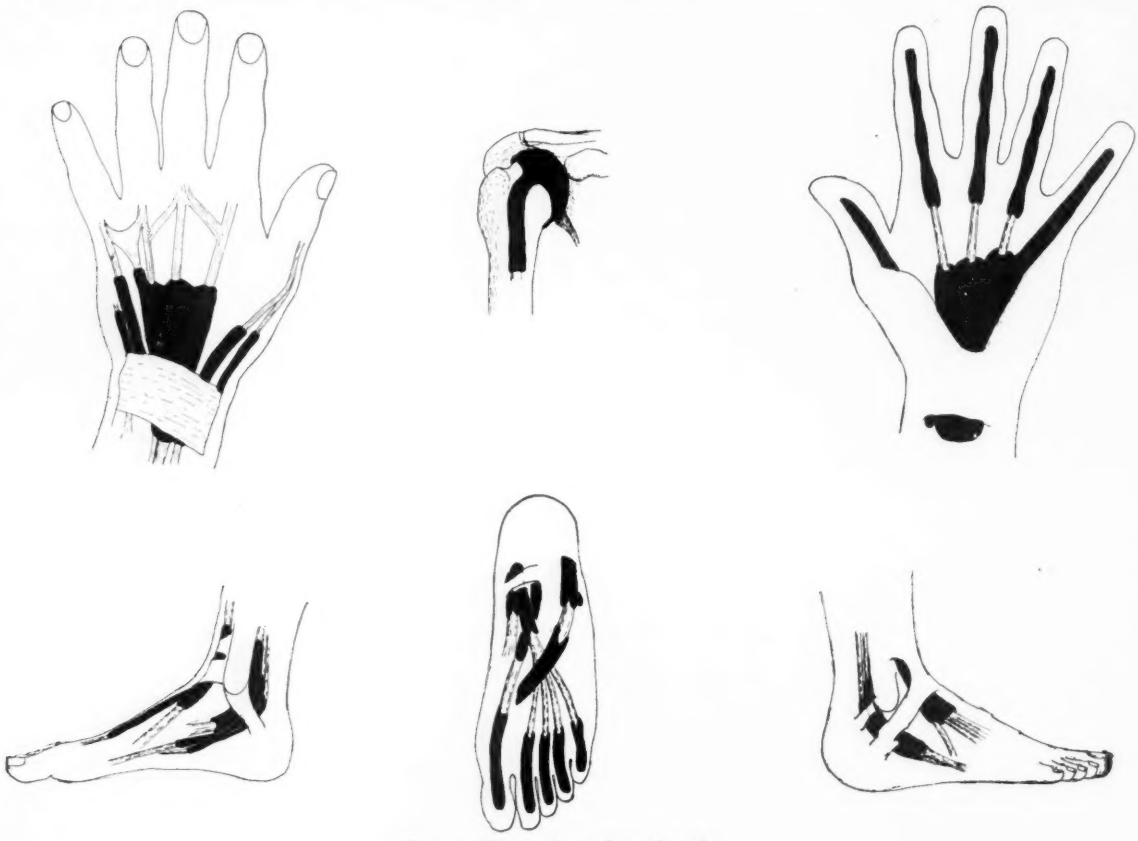

Fig. 1. Sites of tendon sheaths.

Fourth: In order to give the desired freedom of motion to the tendon, the sheath extends beyond the retentive ligament for about an inch.[2]

Fifth: When two or more tendons closely parallel one another, a communal sheath or communicating sheaths frequently developed. At the shoulder the synovial sheath merges with the synovial lining of the joint.[2,4]

Sixth: As the range of motion increases, the mesotendon becomes more tenuous and in some cases disappears entirely, leaving only some villous remnants.[2]

Seventh: The synovial lining of joints, bursa, and tendon sheaths are identical histologically and is described by Keith[3] as modified cartilage cells; by Kingsley[6] as epithelial; by Piersol[7] as endothelial. Morton,[8] it seems to me, has most accurately described the tissue when he states, "The synovial cell is a modified mesenchymal cell with multipotential qualities." We may, therefore, anticipate similar diseases affecting joints, bursa and tendon sheaths.

Tendon sheaths are affected by (1) trauma, (2) infections, (3) neoplasms and (4) disturbances of metabolism.

Trauma is probably the most frequent cause of tendon disability and usually appears in the more exposed tendons, viz., the shoulder, the abductor-extensor sheath of the thumb and the peroneal tendons. The injury may be single or multiple, severe or slight. The shoulder usually bears the brunt of the single severe accident while the thumb is usually affected by many small injuries.

Tenosynovitis of the long head of the biceps usually follows falls, or strain of the shoulder, particularly is it likely to follow

a Colles' fracture. It is manifest by pain in the bicipital groove and difficulty in extending, abducting and rotating the

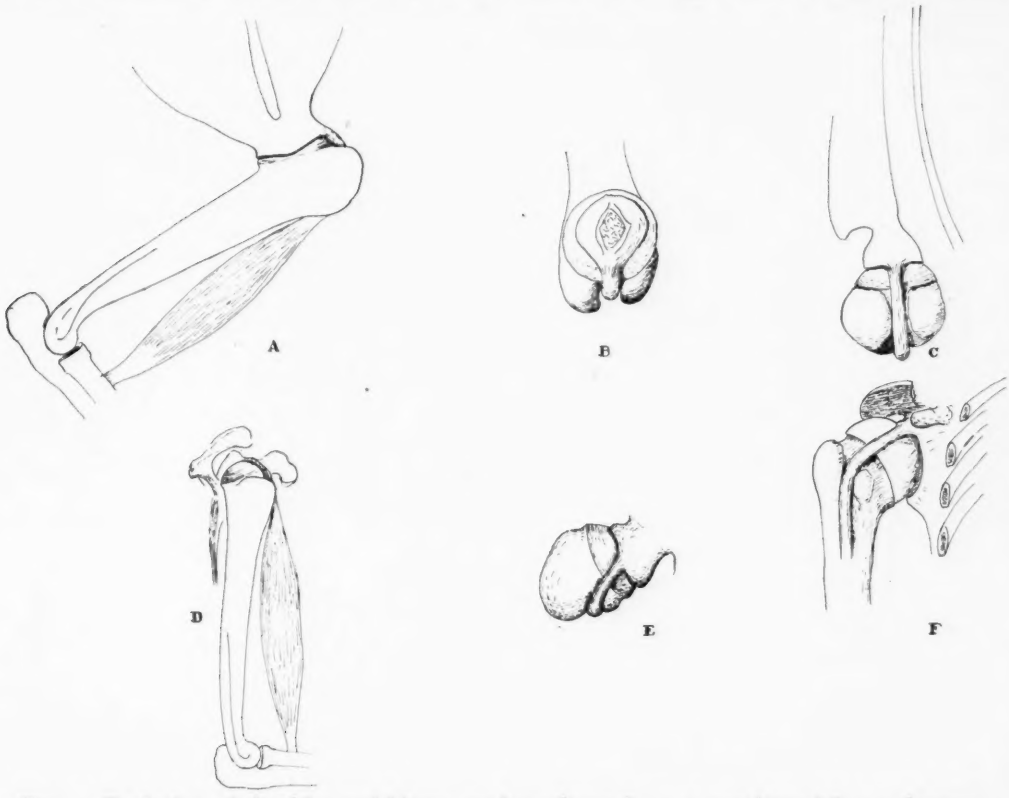

Fig. 2. Evolution of shoulder and biceps tendon-schematic. A–C, quadrupedal; D-F, human.

shoulder. Meyer's[10] careful work has shown the effect of wear and tear at this point, while Abbott[11] has concerned himself with dislocations of the tendon. The common cause of all these troubles is found in the evolution of the shoulder.

With the rise of mammals the legs were drawn close to the body for use in the sagittal plane and quadrupedal posture became habitual rather than occasional. In the quadruped the glenoid faces downward onto the upturned face of the humeral head. (Fig. 2A–C.) The supraglenoid tubercle in many projects beyond the articular surface. The spinati and the subscapular muscles are the chief flexors of the humerus and the development of the tuberosities vary in accordance with the use and weight of the owner. The long head of the biceps tendon runs directly from the supraglenoid tubercle into the intertubercular groove without contacting the articular surface. The forearm of many of these creatures became fixed in pronation or nearly so in order to bring the forefoot on the ground. A cat has about thirty degrees of supination, a dog only a trace. Contraction of the long head of the biceps could, therefore, have only two functions: (1) flexion of the elbow and (2) active support of the humeral head, preventing headward dislocation.

With the rise of the large brachiating primates, including man, changes occurred. The deep and narrow chest became broad and shallow, bringing the scapulas from a lateral to a dorsal position. In their erect position the humerus still parallels the torso but the axis of the articular surface has rotated in respect to the intercondylar axis so that (1) the subscapularis has lost most of its postural and flexor activity, becoming a mere internal rotator of the humerus with a resultant diminution of the lesser tuberosity and (2) the bicipital tendon lies obliquely across the humeral head, causing it to turn sharply around the now recessive lesser tuberosity. Each contraction of the long head of the biceps

would, therefore, tend to draw the tendon against the lesser tuberosity and retentive ligament, causing wear and tear which might eventuate in a fraying or a dislocation of the tendon. Its exposed position would tend to subject it to a crushing injury between the head of the humerus and the acromion. Any motion that would tend to displace the head of the humerus forward would throw additional strain of the bicipital tendon. Such accidents might cause sufficient damage to the tendon producing reparative actions and adhesions causing the characteristic signs of tenosynovitis of this tendon.

Tenosynovitis of the abductor and short extensor of the thumb might almost be classified as a female disease as about 80 per cent of the cases[12] occur in women near the menopause. In various ways they turn their hands into flexion and ulnar deviation as in wringing clothes, throwing the typewriter carriage back into the starting position, etc. Wearing a heavy bracelet would also predispose to this condition. It is reported as an acute temporary condition in males returning to work as carpenters, and gardeners after long layoffs. These slight injuries result in an inflammation of the tendon sheath with early crepitation and subsequently in a scarification of the sheath which gradually constricts the tendon and may cause the appearance of a nodule within the tendon itself. As the tendon is moved either actively or passively, the tendon slips with difficulty through the constriction and a definite snap may occur. At times the tendinous lump may become locked on one side of the constriction. As the condition is frequently occupational in character, it is severely disabling. In one patient upon whom I operated the nodule had white areas that reminded one of the calcareous deposits found at the shoulder and other points. This probably represents an added metabolic disturbance.

Similar conditions, either congenital or acquired, may occur in one or more finger flexors in the region of the metacarpophalangeal joint.

Peroneal tendons are subject to injury when the ankle is sprained by inversion or they may be irritated by spurs from the fibula following sprains. Such an injury, of course, may affect other parts of the foot as well. If the irritation continues, a spasm of the peroneal muscles may be set up which holds the foot in painful eversion. It may be impossible to tell whether the causative factor is a tendovaginitis or arthritis but, fortunately, the treatment is identical.

TREATMENT OF TRAUMATIC TENOSYNOVITIS

The object of treatment in any acute injury should be (1) to permit healing and (2) to avoid disability. Obviously, rest in a position free from strain or contracture is the best method of aiding healing, but rest too long continued is likely to result in adhesive scar formation that will be quite disabling. An injured part, therefore, should be put through the maximal range of motion compatible with retention of the injured parts in close apposition. This does not mean pump handling the part several times a day or once a week, but it means at least moving the part once a day in order to prevent the formation of heavy adhesions.

On the other hand, many of the cases that come to the orthopedic surgeon show that because of pain the part has been kept in one position until the part has "frozen." In the well developed chronic cases of tenosynovitis, two general courses of treatment are open to us depending on the type of lesion.

In tenosynovitis of the shoulder and peroneal tenosynovitis, the demands are such that the part be remobilized by breaking down the adhesions that exist. To accomplish this, the part has to be put through a complete range of motion paying especial attention to the position of the involved tendon.

At the shoulder, the long head of the biceps should be placed in maximum tension with the greatest possible protection that the tuberosities can afford when the part is moved. The first move, therefore,

should be to abduct and rotate the humerus outward about 45 degrees, bringing the tendon directly over the top of the humeral head, obliterating the angle at the lesser tuberosity. The forearm should be kept extended and fully pronated. Maintaining this position, the arm should be carried into full extension and then flexion. The elbow may now be flexed, the forearm supinated and the hand placed between the shoulders in both flexion and extension. Disregarding the biceps tendon, the shoulder may now be internally rotated and flexed and extended through its maximal range in the sagittal plane. Likewise, it should be abducted. Finally, the humerus should be pulled to distract it from the scapula. The hand should then be placed behind the head or neck and the patient should be allowed to awaken in this position as proof of the range of motion.

In frail individuals the arm may be placed in traction in the line of deformity, changing the angle of pull to full abduction as the shoulder loosens up.

The peroneal tendon may be manipulated by maximal inversion of the foot in both flexion and extension. The heel should be raised one-fourth inch along the medial border to prevent the old position from recurring while the patient is standing or walking.

Whatever tendon we are manipulating, it is essential that the individual continue the restored motions of his arm or foot to prevent the reformation of adhesions.

At the crepitating stage the thumb and wrist should be put at rest in a cast, while in a stenosed tenosynovitis of the thumb the sheath must be split or removed in order to give a free range of motion; and if the tendinous nodule is large, it should be excised.

Infectious tenosynovitis is most likely to occur on the palmar surface of exposed hands rather than in the protected feet. Usually it is secondary to wounds but it may be metastatic from remote foci as the urethra, teeth or tonsils and by extension from contiguous bones and joints. Owing to the formation of the tendon sheaths, the infections arising in the little finger and thumb are the dreaded ones because of their direct extension to the carpal area with the possibility of involving all the finger tendons. Infections of the three central fingers are not likely to extend beyond the distal crease of the palm. Infections, too, are serious, not only on account of the plastic exudate that binds them down, but because they are very likely to interfere with the blood supply of the tendons with a resultant interminable necrosis.

Clinically, three major types of infections occur: (1) staphylococcic and streptococcic, (2) gonococcic and (3) tuberculosis.

Streptococcic and staphylococcic tenosynovitis is a worker's disease arising shortly after the receipt of a finger wound. Severe local and general symptoms quickly develop. The sheath and its communicants swell and acute throbbing pain appears with generalized swelling of the hand. Tenderness is exquisite. Pus may be secured early and permanent disability is minimized by prompt treatment. Streptococcic infections are likely to show lymphangitic red streaks up the forearm, a danger signal of the first magnitude.

Gonococcal infections arise metastatically in any tenovaginal sheath of the hot and foolish. The local and constitutional symptoms belie the duration of the infection by their mildness. Aspiration yields a characteristic greasy, gray translucent pus which should be smeared and cultured to confirm the diagnosis.

Tuberculosis is usually secondary to contiguous or remote foci. An insidious onset of tenovaginal swelling in young adults may be the only sign. Tuberculosis elsewhere should make the diagnostician alert. This condition may also be confused with some tumor formations, owing to its tendency to produce small translucent cartilaginous "rice bodies" which may be felt, imparting a peculiar sensation to the examining hand when the part is moved.

TREATMENT OF INFECTIOUS TENOSYNOVITIS

Treatment of infectious tenosynovitis varies with the type of infection. In the

acute staphylococcic type when pus is present drainage is imperative; while in either type, but especially the streptococcic, if lymphangitis is present, no surgery should be done until the lymphangitis is cleared up by hot packs and the use of specific drugs. To do radical surgery in the face of the red flag of lymphangitis is likely to hasten the demise of an otherwise useful citizen. When the lymphangitis has subsided leaving a collection of pus or when pus exists without lymphangitis, the indication is for surgery of both a radical and exact nature. Kanavel[14] pointed out in his monograph the value of certain incisions which (1) kept the scars off bearing surfaces and (2) preserved the retentive ligaments which act as pulleys for the tendons so that the ligaments bend normally and not with a tendinous band extending chord-like between the joints on either side of the one operated upon, resembling the chord of a Dupuytren's contracture. The value of the pulleys cannot be overemphasized. To this end the incision in the fingers should be located posterolaterally along the sides of the phalanges but not crossing the joints. If the thumb or little finger is involved, the sheath may be opened by an incision along the mesial border of the respective eminences, being very careful to preserve the nerve supply of the eminence. If the carpal sheath is involved the incision should be on either side of the wrist just on the palmar surface of the bone. (3) In draining the sheath no foreign body should bear against the tendon, but the sheath should be kept open by vaseline packs. (4) The use of hot antiseptic baths or saline baths tend to reduce the pus and promote drainage. (5) The pus should be examined, the organism identified and the proper sulfonamide drug should be adequately administered. (6) For comfort's sake the hand should be splinted in nearly full extension to prevent contractures and (7) the hand should be elevated.

Gonorrheal tenosynovitis should be treated by the use of proper drugs, splint and heat, but under *no* circumstances should the sheath be opened and drainage established.

Tuberculous tenosynovitis demands a complete, careful dissection of the diseased area which may necessitate reconstructive measures later. The removal of the finger flexors might call for a fusion of the wrist and muscle transplants to the remains of the flexor tendons. Potts[13] advises *x*-ray treatments twenty-four hours in advance of surgery, and again a week after surgery he believes that *x*-ray treatments should begin and be repeated every two weeks. Personally, I have had no experience with this physical adjuvant. In the presence of severely destructive lesions and old sinuses one should consider amputation as the shortest means of rehabilitation.

SUMMARY

Tenosynovitis of two types has been discussed and the common traumatic and the rarer infections have been described. Both are severely disabling, require exact diagnosis and prompt, proper care for their alleviation.

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TRAUMATIC VASOSPASM AND ITS RELATIONSHIP TO WOUNDS OF THE LOWER EXTREMITIES*

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THE casualties of modern war as well as the increased number of industrial accidents in defense-booming mills and mines have stimulated renewed interest in the problem of wound healing.

Gas gangrene, particularly in wounds of the lower extremities, is one of the most dreaded complications of compound fractures. The struggle for its prevention, as well as for the control of other pyogenic infections of the soft tissues, of osteomyelitis and of delayed and nonunion, has long called forth the best efforts of the finest medical minds. It is, therefore, with some reluctance, and only in view of existing world conditions, that I present the following conclusions at this time. Should this seem a premature report, I offer in mitigation the urgency of the moment and the hope that those interested in this field may receive it with sympathy and give it further study.

It is my belief that lumbar sympathetic nerve block has a definite place in the routine treatment of compound fractures and severe wounds involving the lower extremities.

This conclusion is based on the reasonable supposition that any force sufficient to damage severely the tissues or to fracture heavy bones is sufficient to disturb the blood vessels, directly or indirectly, by damaging the sympathetic nervous system and thus producing arterial spasms of varying degrees. Thus, I am convinced that many patients treated in the emergency rooms of large hospitals for compound fractures and other extensive wound damage to the extremities also suffer *some degree* of traumatic vasospasm. Because of the importance of the visible injury, which may easily monopolize the attention of the physician at the time, such vasospasms have in the past gone unrecognized.

My interest in the problem of wound infection in traumatic cases has extended over a number of years. From 1927 to 1936 at the Mercy Hospital a great number of such cases came to my attention; the majority were men who worked in coal mines. The incidence of gas gangrene among these injured miners was high, despite careful and studied attempts to prevent infection. More than fifty such cases came to my attention in that period. Our conclusion then was that best results could be obtained by early treatment of the wounds, careful débridement, immediate fixation of fragments and immobilization of the part to prevent further destruction of muscle by sharp edges of the fractured bones. It was found that if the wounds were left open and packed loosely with gauze, infection was less likely to occur, and that when it did, it was less severe.

Yet, despite these steps and an alertness to improve the technic whenever and wherever possible, it was obvious that the great problem of preventing infection was unsolved. The incidence of infection was still far too high for complacency.

Twelve years of work in my clinic for vascular diseases of the lower extremities has made me acutely conscious of the various circulatory problems which confront the surgeon. It has been my experi-

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ence to see many severe cases of vascular spasm—several the result of direct trauma to large vessels.

It was not, however, until one day when called upon to treat a patient suffering from a compound fracture of the tibia and fibula in a hospital of a nearby town that the importance of traumatic vasospasm in relation to traumatic wounds of the legs was clearly brought home to me.

Case I. I first saw the patient, a young adult, several hours after the accident. He lay upon the operating table of the emergency room, suffering from a compound fracture of the tibia and fibula, with a large gaping wound extending from the knee to the ankle. All muscles were evulsed and many structures exposed. The bones protruded from the wound. The skin was pale and the tissues had a cold and clammy feel. The femoral pulsations were present but faint. The popliteal pulsations were absent. There was no dorsalis pedis pulse. The general condition of the patient was fair; the degree of shock was slight.

After an intravenous injection of glucose and saline, the patient was removed to the operating room. Sodium pentathol anesthesia was administered. After careful débridement of the destroyed tissue, the fractured bones were approximated and plated. The wound was packed and loosely closed. Lateral plaster splints were applied to the leg.

The patient was then turned over, face down, and a paravertebral lumbar sympathetic block was performed with 20 cc. of 2 per cent novocaine. He was then sent to bed.

Within about ten minutes, although the exact time was not noted, the toes were quite pink, and the femoral pulse was much more vigorous. (The popliteal pulse could not be felt because of the plaster splints.)

The patient made an entirely uneventful recovery. The fracture united in the normal length of time and the wound healed without complication.

In this case the traumatic vasospasm was obvious. However, the relationship of the spasm to the wound, opened up to me a new line of thought.

Was it not reasonable to suppose that in all serious wounds of the lower extremities there is some degree of spasm, which, by diminishing the blood supply to the part, gives the infecting organisms time to establish themselves firmly? Such unrecognized vasospasms might be the reason why compound fractures of the extremities are so likely to become infected. As Dr. Griffith[1] points out, there is no better therapeutic "chemistry" to be introduced into a wound than that which nature produces from clean muscles and an adequate blood supply. Any procedure, within the realm of safety, which would increase the blood supply to the injured part would be justified.

By blocking the sympathetic ganglia with novocaine, the vasomotor reflex would be broken, the contraction of the artery released and the flow of blood to the part greatly accelerated.

The technic of the lumbar sympathetic block is comparatively simple, and if done carefully can cause no possible damage to the patient. Ochsner and DeBakey[2] perform it regularly on out-patients when they wish to differentiate between arterial deficiencies due to organic disease and those due to vasospastic states. They point out that chemical block of the regional sympathetic ganglia is a conservative procedure in peripheral vascular disease because of its efficacy, its simplicity and its facility of performance.

I was so stimulated by the results of the nerve block in the case described that when, less than a month later, a patient with soil-contaminated compound fractures of both legs was admitted to my service I decided again to use the nerve block.

Case II. The danger of infection in this case was clear since the patient, a boy seventeen years old, employed in a slaughter-house, had been run over by a truck in the slaughter-house yard. Not only had his clothing been contaminated by the feces of the animals with which he worked, but portions of it had been ground into the wounds by the wheels of the truck.

The patient's left extremity suffered an extensive wound exposing large groups of

muscle. The fracture was compound comminuted and a fragment of bone had been lost. A compound fracture of the right leg was exposed by a wound less severe than the left. Both the limbs, as in the first case, were cold and clammy out of all proportion to the mild degree of shock which existed. The pulsations of the femoral vessels were weak and the popliteals and dorsalis pedes were not palpable.

This lad was taken to the operating room immediately. Both wounds were carefully débrided and sulfathiazole was powdered over the extensive wound on the left leg, while none was used upon the wound of the right leg. The bones of the left leg were approximated and a Sherman bone plate used to fix them in position. The fractured portions of the bone in the right leg were approximated but no plate applied. Both wounds were lightly packed with gauze and the wound edges loosely approximated. Lateral plaster splints were applied to both legs and in this instance the application of the splints was followed by a bilateral lumbar sympathetic block.

This boy made an uneventful recovery. The wounds healed without any evidence of infection of soft tissue or bone. The boy left the hospital in 120 days and five months afterward was able to walk without the aid of a crutch or cane.

Case III. A healthy young adult had fallen thirty feet from a crane he was operating in a steel mill. He suffered an extensive compound fracture involving the ankle joint. He was treated in precisely the same manner as in Case II namely, a careful débridement within an hour after the accident, reduction of the fracture, loose closure of the wound, packing with dry gauze, application of plaster splints and a lumbar sympathetic block. This patient also made an uneventful recovery without infection.

Several other compound fracture cases after these, although showing no marked evidence of arterial spasm, seemed to benefit by the paravertebral sympathetic block of the ganglia.

It is a well recognized fact, as noted by both Kroh[3] and Homans,[4] that traumatic vasospasm is a definite entity. This vasospasm can exist, they point out, because of direct trauma to the vessels, or in many instances, as the result of disturbances to the local vasomotor nerves which influence the functioning of these vessels. Perhaps the cause makes little difference, since, in any case, a nervous mechanism in close relation to the vessel seems to have been set in motion.

Homans[4] also points out that traumatic arterial spasm may be brought on by a variety of injuries, including fractures, blows, bullet wounds and stabs; and that although there is sometimes evidence of direct trauma to the arterial wall, almost as often the artery itself seems never to have been touched.

Cases of acute arterial spasm resulting from fractures have been described by Montgomery and Ireland[5] and in these it was impossible to state whether the spasm was induced by direct trauma to the artery or whether by injury to the artery's sympathetic nerve supply.

It seems reasonable to contend that any force sufficient to damage severely the tissues or to fracture heavy bones is sufficient to disturb the blood vessels, directly or indirectly, by damaging the sympathetic nervous system which controls the size of the lumen and thus to produce arterial spasms of varying degrees, diminishing the blood supply at the very time it is needed for healing of the wound.

Spasms due to trauma last for hours, even for days. It is true that in a limb uncomplicated by an open lesion or a fracture, there is as a rule, no complicated aftereffects from a traumatic vasospasm, although I have seen cases in which several toes became gangrenous and were lost as a result of this type of injury.

In compound fractures of the lower extremities, it appears that the vasospasm is a transitory thing and usually passes off itself in due time. However, the period of time necessary for nature automatically to release this spasm gives the infecting organisms time to establish themselves firmly. The generalized ischemia set up by the spasm promotes an excellent medium for bacterial growth. The nerve block

shortens the dangerous interim of diminished blood supply by releasing a rich flow of blood to the injured part at this critical time.

Brown[6] has stated that wounds about the face involving bones that do not communicate with the oral cavity rarely become infected. He attributes this rapid healing of bone in this region to the excellent blood supply. Compound fractures involving the arm, while they may become infected, do not present the same high incidence of infection as those of the legs; and again this may be attributed to a better blood supply in that region.

It seems to me, after observing and treating many compound fractures of the extremities, that release of the vasoconstrictor impulses of the accompanying but usually unrecognized vasospasm may mean the difference between the successful healing of wound and infection. I believe that many cases of osteomyelitis, gas gangrene and other pyogenic infections of the soft tissues and possibly also of delayed and nonunion, could have been prevented had we recognized the importance of traumatic vasospasm in its varying degrees of intensity in the extremities of those treated for serious wounds and compound fractures. Although the number of cases in this report is not great, the results are so gratifying as to indicate that the lumbar sympathetic nerve block has a definite place in effective therapeusis in compound fracture and severe wounds of the lower extremities.

TREATMENT

I suggest that adequate treatment of compound fractures of the lower extremities is as follows:

1. Early primary attention and all possible speed in getting the patient to the hospital. As Kennedy[7] states, "the length of time between the occurrence of the injury and the accomplishment of débridement is of almost equal importance with the method employed." Satisfactory healing depends upon the treatment given a wound in the first few hours. There should be careful splinting at the scene of the injury by the doctor in charge or by the first aid man. The best surgeons should be in the casualty clearing stations and not behind the lines, in the emergency rooms of hospitals and not beyond immediate call. Gone are the days when initial treatment consisted of a resident physician putting a sterile dressing on the patient and sending him to bed to await the morning call of the physician in charge.

2. Careful evaluation of the patient's general condition and an estimate of the degree of shock.

3. If the patient's condition permits, he should be taken to the operating room where, under the best available conditions, all destroyed tissue and extraneous materials should be painstakingly removed from the wound. The fracture should be approximated with as little trauma as possible. We believe that a bone plate should be used to fix the fragments in position unless there is such digitation of the fracture ends that they can be approximated with a reasonable assurance that they can maintain their position.

4. At this point antiseptic drugs such as sulfathiazole may be sprinkled into the wound. I, personally, use sulfathiazole, although, as indicated in Cases I and II, in which one extremity served as a control, wounds seem to heal as well without it in compound fractures when the sympathetic nerve block is used. Also, although in treatment of open wounds the use of polyvalent anaerobic vaccines is common, I believe that they have no great importance and do not use them.

5. The wound should be very loosely closed and drainage insured by a packing of loose gauze. Permitting the air to enter the wound lessens the chances of anaerobic growth.

6. Either a moulded lateral plaster splint or a solid cast should be applied. I favor splints as they enable the surgeon to examine the wound at will and do not necessitate the cutting of a window in the plaster cast when such examination is

necessary. Also, as inflammation and swelling subsides, these lateral splints may be tightened and so maintain more complete fixation of the limb. As Eliason[8] points out, "a leg confined in a cast, after the swelling has subsided, very often rattles like a pea in a pod."

7. The patient should then be turned on his side (the side opposite the fracture) and a novocaine block of the lumbar sympathetic ganglia performed. In cases of deep anesthesia, it is permissible to turn the patient upon his face. We feel that the vasospasm in compound fractures is a transient one and that 20 cc. of 2 per cent novocaine is sufficient to give the results sought.

Technic of Lumbar Injection. A wheal is made about 7 cm. from the midline opposite the second lumbar interspace. This falls close to the twelfth rib. Through this wheal more novocaine is injected into the muscular aponeurosis which is quite sensitive. A lumbar puncture needle or a No. 22 gauge needle should next be used. This is introduced at a right angle. Often it meets the tip of a transverse process. If so, it can be withdrawn and reinserted at a 45° angle toward the vertebral body. If it strikes the body, it should be partially withdrawn and adjusted so as to pass the body completely for a distance of about two and one-half inches. There it should lie on the psoas muscle, just behind the aorta or inferior vena cava. At this point extreme care and caution should be used as it is possible to enter the vena cava. A careful pulling back of the plunger of the syringe will tell if it occurs. If this occurs (and it has happened to us), no difficulty follows. Twenty cc. of 2 per cent novocaine are infiltrated into the tissues at this point, which in itself should suffice. To be doubly sure, it is well to carry out the same procedure in the third interspace. In several of our cases I have injected 5 to 10 cc. of 90 per cent alcohol with no results other than which we wished to obtain.

SUMMARY

1. The theory is advanced that some degree of traumatic vasospasm is suffered in all cases of compound fracture or serious wounds of the lower extremities.

2. This theory is based on the supposition that any force sufficient to damage severely the tissues or to fracture heavy bones is sufficient to disturb the blood vessels, directly or indirectly, by damaging the sympathetic nervous system and thus producing arterial spasms of varying degrees.

3. Although the vasospasm passes off in due time, it is postulated that this period of diminished blood supply to the part gives the infecting organisms time to establish themselves.

4. A lumbar sympathetic nerve block, to prevent the interim of diminished blood supply, is advocated as routine in the effective therapeusis of compound fractures and severe wounds of the lower extremities.

5. Several cases of severe compound fracture in which the nerve block was used with highly satisfactory results are described.

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SEVERE ACUTE INJURIES OF THE KNEE

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CONSIDERING the frequency of severe acute injuries of the knee joint other than fractures and dislocations, the dearth of literature on this subject is rather surprising. McMurray in his essay "The Diagnosis of Internal Derangement of the Knee" states, "Since no joint in the body is liable to such a multitude of severe or trivial injuries as is the knee joint, a clear understanding of the relative values of the various signs and symptoms is essential if the correct line of treatment is to be carried out." Certainly there can be no difference of opinion in regard to this statement and yet there are only comparatively few articles on the pathology, symptoms and diagnosis of the acute trauma. Most writers have dealt with those conditions which are the result of the severe acute injuries such as the internal derangements and the persistent laxity of the ligaments which have resulted in varying degrees of instability of the joint and they have suggested certain surgical procedures for the correction of these conditions.

In that so many of these cases do come to operation, a most likely conclusion would be that the damage done at the time of the initial injury is irreparable without the aid of surgery. But in our own experience and from the reports of others even with subsequent operations the results obtained in a large number of these cases fall far short of those desired in restoring the normal function of the joint. It would seem, therefore, that the greatest possibility in improving our results in these knee cases lies in the early and accurate diagnosis of the initial injury and instituting the proper treatment at that time. This certainly is a problem of no little economic importance.

There is little wonder that injuries of the knee are so common when the structure and mechanism of the joint is considered. The knee is mechanically a weak joint, being placed between the two longest bony levers in the body, and dependent on its ligaments and supporting muscles for its strength rather than on any particular bony structure. The knee is more than a hinge joint having a definite rotation of the tibia on the femur at the end of complete extension. In addition to this in most occupations and activities when leverage is exerted on the joint it is supporting the body weight and it is at this time that the damage takes place. The range of normal motion of the joint is limited only by the ligaments, and if the joint is carried beyond this, these inelastic structures are obliged to suffer the primary damage and that to the other anatomical structures becomes secondary.

In this paper we have studied the series of 587 cases of severe acute injuries which have occurred in our private practice in the last twenty years. These cover a fairly large percentage of injuries occurring in athletes and industrial workers. From this clinical study we have drawn conclusions which may not be in accord with other observers; however, we believe they are accurate. Along with these observations we have carried out experimental work to support our conclusions and to prove the rational of the treatment suggested although we claim no priority in many features of this.

Briefly our conclusions are: (1) The primary damage is usually to the ligamentous structures and the medial collateral and crucials are the structures most frequently damaged. The damage to the semilunar cartilages are secondary. (2) The effusion in a traumatized knee is practically always due to hemorrhage if the swelling takes place immediately. (3) The disten-

tion of the joint by this blood is one of the greatest factors in producing an unstable joint as well as the hypermotile cartilages with the subsequent locking and damage to these structures. (4) That efficient treatment of an injury to the ligaments must: (a) prevent the prolonged distention of the capsule; (b) protect the injured ligaments, and (c) avoid the atrophy of disuse and maintain the muscular tone of the supporting muscles especially the quadriceps and vasti.

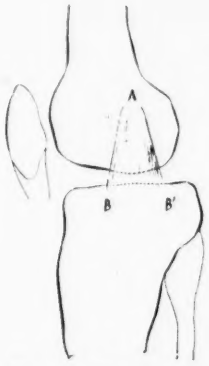

Fig. 1. Shows that the whole ligament is tense in extension.

Fig. 2. When knee is flexed, posterior portion of ligament is relaxed.

Injury to the Internal Lateral Ligaments

In the series of 587 cases a diagnosis of tear of the internal lateral ligament with or without crucial ligament injury was made in 416 cases or approximately 71 per cent of the total. The diagnosis was made on the history of a strain or twist, followed by effusion and tenderness over the head of the tibia at the attachment of the ligament (87 per cent) or over the attachment of the condyle of the femur (13 per cent) and pain, with or without instability, on abduction or external rotation of the leg when the knee is straight. Such findings, when the radiographic examination is negative, justify a diagnosis of injury to this structure.

The internal lateral ligament, the most often injured structure, is one of the most important factors in maintaining the stability of the joint. It is thick and narrow above at its attachment to the internal condyle of the femur but spreads out fan-like below and is attached to the inner aspect of the head of the tibia. When the knee is extended, the internal lateral ligament lies exactly over the middle of the inner side of the joint and all of its fibers are tense. However, when the joint is flexed, because of the inward rotation of the head of the tibia, the whole ligament is carried nearer the posterior aspect of the joint and the posterior fibers become relaxed. (Figs. 1 and 2.) If in this position (semiflexion) abduction of the leg is attempted, the strain is thrown on the tense anterior portion of the ligament. This can be easily demonstrated on the freshly dissected knee. The external rotation of the tibia on the femur is limited by the same fibers of the ligament. The internal semilunar cartilage is adherent to the deep fibers of the ligament except for its anterior cornu which is bound to the head of the tibia by that thinned part of the capsule spoken of as the coronary ligament. Both menisci act by their wedge shape in adapting the surface of the head of the tibia to the condyles of the femur in the varying positions. Thus it will be seen that the prevention of abduction in the extended position is due to the whole internal lateral ligament, but that abduction and external rotation are limited only by the anterior fibers of the same ligament when the knee

is partially flexed and before any strain is thrown on the posterior fibers the anterior must give away.

It is true that the crucials also prevent abduction in the extended position, but in the partially flexed position these are relaxed and again the tense anterior fibers of the lateral ligament must give away before the strain falls on the crucials.

The extensor muscles, especially the quadriceps and vastus internus by their fibrous expansion over the inner side of the capsule when they are contracted, are worthy reinforcements in resisting abduction. It is needless to call attention to their importance as stabilizers in the extended position and to a lesser degree even in slight flexion.

Without argument it seems fair to assume that the majority of injuries to the knee takes place with the joint slightly flexed and the traumatizing force carrying the leg into a position of abduction and external rotation. This is almost universally accepted in the literature on the subject and the histories of our cases substantiate this: when sufficient force is exerted in abduction or external rotation, the anterior fibers of the lateral ligament are first to give away. The ligament does not usually tear in the middle from such force but practically always ruptures at or near the bony attachment. In a series of experiments carried out on the knee joints of cadavers we have never seen the internal lateral ligament rupture in its middle but always from its attachment and usually from the tibial head and rarely from the femoral condyle.

In the cadaver experiments ten knee joints were used. All of the muscles about the joints were divided and the inner side of the capsule exposed. The abducting force was applied to the joint with the knee extended in five cases. Three of these gave way from the tibial head, one from the femoral condyle and one from both the condyle and head of tibia. In the five joints in which the abducting force was applied with the knee in slight flexion all showed that the anterior fibers of the ligament gave away first from the tibial head. The same experiments carried out on living dogs were not as convincing as to the weakest part of the ligament. In both the flexed and extended position more than half pulled away from the femoral condyle. We think this is explained, however, by the different shape of the internal condyle in dogs and the fact that the attachment to the femur is as broad as it is below at the attachment to the tibia and there is no difference in the relaxation of the anterior and posterior fibers when the knee is flexed.

Because of the intimate connection between the semilunar cartilage and deep fibers of the ligament, any force which pulls the attachment of the former away from the head of the tibia must lift the cartilage away from the bone also. It is then evident in that the anterior fibers of the ligament are most frequently ruptured so that the anterior part of the cartilage is most frequently displaced from its tibial head. This coincides with the view of S. Alwyn Smith that "a displacement of the internal semi-lunar cartilage cannot occur unless the internal ligament be stretched or ruptured." In over two-thirds of our cases of internal ligament damage demonstrable abduction was present when the knee was extended.

Hemorrhage into the joint was a common finding in the 416 cases. In the whole series of knee injuries 93 per cent showed macroscopic blood on aspiration. These figures agree with those published by Harding in 1919. This effusion is a factor of the greatest importance in preventing the proper healing of the ligament and cartilage in their proper relation to the head of the tibia. (Figs. 3 and 4.) It usually takes from one to three weeks for the effusion to be absorbed under rest and bandaging during which time the torn ends of the ligament and the cartilage are held away from the head of the tibia. While this is the case, the interval between these structures is bridged across by granulation and young cicatricial tissue which fails to fix the

cartilage closely to the head of the tibia and actually lengthens the ligament. This must result in a loss of stability of the joint.

Fig. 3. Shows how distention of joint separates torn ends of internal lateral ligament.

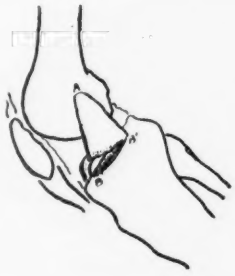

Fig. 4. Shows torn anterior portion of ligament most widely separated by distention.

For the purpose of demonstrating the effect of the effusion in separating the cartilage from the head of the bone, the knee joints of a fresh cadaver were subjected to abduction and external rotation while the knee was semiflexed until the internal lateral ligament could be felt to give away. The joints were then opened through a split patella incision and the interior of the joints examined. The internal cartilages were found to be lying smoothly in their place on the tibial head. The anterior part of the cartilages were then covered with silver foil which was sutured to the cartilage. The joints were closed and sealed with collodin. *X*-rays taken at that time showed the shadows in their apparent normal position. (Fig. 5.) The joints were then distended by means of a needle and syringe, as much as 100 cc. of fluid being used. Plates then taken in exactly the same position showed the cartilage shadows had been lifted away from the tibial head. (Fig. 6.) This explains the *x*-ray appearance when the joint is distended with oxygen and the gas shadows show under the cartilage when it is detached.

This would indicate what might be expected in a certain number of our patients treated by simple rest and splinting, a hypermobile cartilage and an inefficient ligament if no other factors were present. We have already stated that although the

Fig. 5. Shows internal semilunar cartilage in normal position on head of tibia before the joint was distended.

Fig. 6. Shows how cartilage has been pulled away from the head of the tibia when the joint was distended.

muscles of the thigh are not the real factors in preventing lateral mobility of the knee, they are contributing factors and must be taken into account. It is true that these are seldom injured at the time of the sprain but the disuse which must follow rest or splinting results in weakness and atrophy. It has been suggested that the atrophy of the vastus internus in traumatized knees is greater than that of simple disuse. The explanation offered for this is that this muscle is supplied by the same nerve as is the joint. However, by careful observation and measurements in a number of our patients who were splinted at rest for several weeks we have been unable to note any difference in the degree of atrophy compared with those which have been fixed for a similar period for other conditions such as a fractured foot or leg.

In the foregoing we have tried to emphasize that there are three factors of paramount importance in the treatment of the acute injuries of the lateral ligament and internal meniscus:

1. *Prevention of Distention of the Capsule.* Paracentesis has been advised by many for the relief of pain when the disten-

tion is great. Harding and McWilliams have reported excellent results following early aspiration of traumatized joints. The

Fig. 7. Shows hinges used in hinged cast.

maximum degree of distention takes place within the first thirty-six hours after the initial injury and this seems to be the most favorable time for aspiration. Our experience has been similar to Metcalf, that is, when aspiration is performed earlier than twenty-four hours the effusion has usually recurred which necessitated a second aspiration. The amount of fluid removed has varied from 30 cc. in the mildly distended joint to 120 cc. in the very tense joints. Following the aspiration the joint is almost entirely relieved of pain and the simple motions of flexion and extension of the joint become free and painless. In nine cases in which the knee was locked and difficulty experienced in reducing the apparently dislocated cartilage, aspiration was done immediately and simple manipulation accomplished the reduction. Whether the distended capsule was the factor in holding the cartilage out of place we cannot say, but the ease of reduction after aspiration was certainly suggestive.

2. *Protection of the Injured Ligaments from All Strain.* Immediately following aspiration a light plaster cast is applied in two sections joined by a pair of special hinges placed on both sides of the joint which allow flexion and extension of the knee but which prevents all lateral movements or rotation. This allows movement of the knee but protects the injured ligaments.

Our method of application has proved very simple and effective. The hinges are made by the brace maker and are inexpensive. (Fig. 7.) Four strips of adhesive plaster one and one-half inches wide and slightly longer than the section of cast are used. These are applied to the front, back, inside and outside of the thigh so that there will be free ends above and below the cast to be turned over the edge of the plaster and incorporated in it. (Fig. 8.) Without padding several layers of plaster of Paris are applied and the ends of the adhesive are turned over the upper and lower edges and incorporated by covering these with additional layers of the plaster. A similar cast is then applied from the head of the tibia to just above the ankle, a pad of sheet wadding having covered the front of the tibia. After these sections have hardened, the section on the thigh is rotated outward as far as possible and the one on the leg rotated inward as far as possible. While they are held in this position the hinges are applied to the sides of the knee and fixed to the two sections by several additional layers of plaster bandages. (Fig. 9.) After the sections have set they are trimmed around the knee sufficiently both in front and behind the joint so that full extension and flexion to at least 90° is possible. (Figure 10 shows the complete cast and Figure 11 the range of motions possible.) It is to be noticed that the knee can be readily observed and is easily accessible for further aspirations if necessary.

These patients are kept quiet for forty-eight hours after the aspiration, and if no recurrence of the effusion takes place, weight bearing is allowed on the third day. On the fifth or sixth day usually the patients are getting about comfortably with only the use of a cane which they soon discard. As an additional protection to take

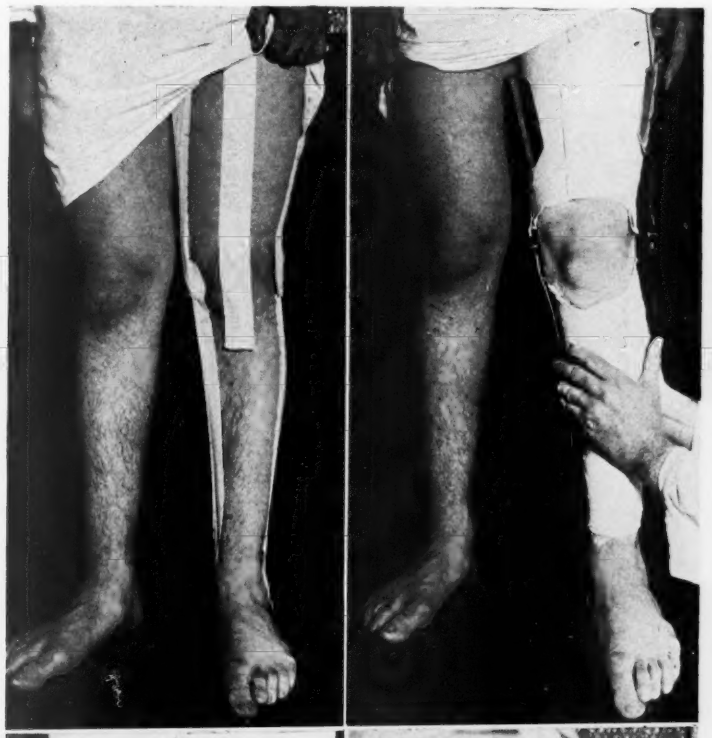

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 11.

Fig. 8. Shows adhesive strips applied to thigh and calf before plaster is applied.

Fig. 9. Shows the thigh and leg cast joined by the hinges.

Fig. 10. Shows the completed cast properly trimmed.

Fig. 11. Shows range of flexion.

the strain off the inner side of the joint the heel of the shoe is elevated one-fourth inch on the inner side.

3. *Avoidance of Atrophy and Maintainance of the Muscular Tone of the Quadriceps and the Vasti.* The necessity of maintaining the muscular power has been stressed by many writers and the importance of exercise and massage emphasized. We believe that the use of the muscles in walking is the best exercise and the improvement of the local circulation by such exercise is of decided advantage. This, however, should be carried out only if the proper protection is afforded the injured structures and the cast splint seems to furnish this. Weight bearing and use of the joint is begun as early as the third day after the aspiration and are supplemented by special exercises for the extensor muscles. These simple exercises are carried out by having the patient extend the knee actively and fully while sitting. He is instructed to carry this out for at least fifty times thrice daily. These are carried out for the six weeks that the cast splint is worn and for several months after its removal. We have been impressed with the moderate atrophy which takes place in patients so treated. When the cast is properly applied and not made heavy, it is surprising how well these patients walk and are often able to get back to light work in a week or ten days.

Several years ago a smaller group of patients with internal lateral ligament damage (this group is included in the present series) who were treated by the method outlined were studied in comparison with a group of similar patients treated by cast or splint and followed by compression bandages or strapping. The results are given in Table 1.

TABLE 1

247 cases of severe acute injuries of the knee

168 cases (68 per cent) diagnosed as injury of the internal lateral ligament and semilunar cartilage including 32 cases of rupture of the crucial ligament (anterior) and 9 cases of fracture of tibial spine

146 cases (87 per cent) showed macroscopic blood on aspiration

Of the 168 cases, 88 cases were observed over 3 years or more.

	Recurrent Attacks		History of Locking Subsequently		Cartilage Removed	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Treated by aspiration and hinged cast, 62	11	16	3	5	2	3.2
Treated by cast or posterior splint and bandaging, 26	15	60	6	23	5	19

	Treated by Aspiration and Hinged Cast	Recurrent Trouble	Treated by Cast, Etc.	Recurrent Trouble
Crucial ligament complication	7	2	4	2
Fracture tibial spine	4	0	3	1

INJURY TO THE EXTERNAL LATERAL LIGAMENT

Injury to the external lateral ligament has been comparatively rare as a single lesion, the diagnosis having been made only twenty-one times in the series. Of course in the most severely injured joints it unquestionably has occurred along with the rupture of the other ligaments when the knee is unstable in all directions. The diagnosis of rupture of the external ligament has been based on the history of an adducting trauma effusion, tenderness over its attachments to the external condyle or the head of the fibula, pain on adduction of the leg on the femur and abnormal adduction mobility. We do not believe that injury to the external cartilage is related to the injury to the external ligament as the cartilage is not attached to it, having the popleteus muscle interposed between these structures. The treatment as suggested for injury of the internal lateral ligament is recommended with the exception, of course, of elevation of the inner side of the heel.

RUPTURE OF THE CRUCIAL LIGAMENTS

Injuries and rupture of the crucial ligaments have been grouped with those of

injury to the internal lateral ligament as rupture of the crucials cannot take place without damage to the medial collateral. Pringle has produced rupture of the anterior crucial by flexion abduction and internal rotation but in all of his cases the deep fibers of the internal lateral were also torn. Jones and Smith as well as many others are in accord with this view. In addition to the symptoms of an injury to the lateral ligament, if the anterior crucial is torn, there is abnormal forward movement of the tibia or the femur and abnormal internal rotation when the knee is straight. An abnormal backward movement indicates an injured posterior crucial. We had this finding only six times in our whole number of cases and all of these were in joints that had been completely dislocated. Injury of the crucials was made in 26 per cent of the internal lateral group. The same treatment is indicated in these cases as in the latter although the hinged cast should be worn somewhat longer, for eight weeks at least.

SEMILUNAR CARTILAGES INJURIES

We are not prepared to say what percentage of the cases showed meniscus damage at the time of the initial injury. We believe that in practically all of the cases of rupture of the internal lateral ligament from the tibial head the attachment of the cartilage was also torn. But the splitting and laceration of the cartilages is caused by the engaging or pinching between the condyles and tibial head and does not take place until it is traumatized in such a way at least once. That this does occur at times at the initial injury is indicated that the joint immediately locked in fifty-four of our cases. Of course in those cases in which the knee is locked in flexion immediate reduction of the displaced cartilage by manipulation is indicated and also application of the hinged cast made at once. We believe strongly that conservatism should be the rule even in the very severe ligamentous injuries. To operate for a torn or hypermotile cartilage in the acute stage and to ignore the primary damage to the ligaments that accompanies the condition is the height of poor surgical judgment. In exceptional cases, however, when the laceration of the ligaments is so severe that apposition of the torn ends seems impossible, exploration and suture of the torn structures is justifiable. In these cases it is wiser to delay the operation for at least forty-eight hours after the injury. The same protection and care must be carried out postoperatively as in those treated conservatively.

FRACTURES OF THE TIBIAL SPINE

In the series we have encountered twenty-nine cases of fracture of the tibial spine. In practically all of these cases the symptoms of internal lateral ligament injury have been present. In eighteen cases there was demonstrable abnormal forward movement of the tibia on the femur—the so-called rocker sign. The fact that only eighteen out of the twenty-nine cases had this, supports the view of Jones and Smith that fractures of the tibial spine are not always avulsion injuries by the strain on the crucial ligaments but the condition does occur without crucial ligament instability. The diagnosis in all of these cases was made by radiographic findings. This emphasizes again the importance of adequate *x*-ray study in all acute knee injuries. In only nineteen of our cases was there a block to full extension of the joint. If, as has been stated, such blocking is the most constant symptom of fracture of the tibial spine we were unable to demonstrate it in these cases. Most of our cases have been treated by aspiration and fixation in a straight cast for six or eight weeks. In about one-third the hinged cast has been used and in these we believe we really have gotten better results with much less atrophy and general relaxation of the joint than in those in which the straight cast has been used.

BRUISING OF THE INFRAPATELLA FAT PAD

Contusion or bruising of this structure is a definite traumatic entity and is caused

usually by a direct blow on this area of the joint. At other times it is a complication of ligamentous injuries. The diagnosis is made by the swelling and tenderness just below the patella and to either side of its tendon. There is usually a feeling of fullness at this site and pain on full extension of the knee but there is no definite locking or blocking of the joint as seen in the cartilage and tibial spine cases. At times the symptoms do not come on immediately following the trauma but gradually increase for twenty-four or forty-eight hours. In the most acute cases aspiration, if effusion is present, and rest in a plaster cast with the knee flexed to about 20° for at least two weeks is indicated. This should be followed by a compression bandage and an elevation of the heel of the shoe on the affected side for an additional two or three weeks. In the milder cases the latter part of this treatment is all that is needed. The diagnosis of this condition was made thirty-one times in this series.

SIMPLE TRAUMATIC SYNOVITIS

We have been quite cautious in making a diagnosis and have made it only when most careful examination revealed no injury to any other structure except the synovia. It is difficult to see how a sprain or twist can injure the synovia alone but a direct blow or contusion could conceivably injure this membrane and produce effusion (hemorrhage) and general soreness on movements of the joint. In twenty-seven cases we obtained such a history and only with these symptoms did we believe such a diagnosis was justifiable. In the mild cases a compression bandage and restricted activities were all that was necessary. In four cases in which the effusion was marked the joints were aspirated and in all of these the effusion was hemorrhagic.

Unquestionably, there are other pathological conditions often present which are almost impossible of recognition at the time of the initial injury. Probably the best example of such conditions are the injuries which take place to the articular cartilage or the underlying bone (osteochondritis dissicans) or the flakes which are chipped off the semilunar cartilages and later give symptoms of loose bodies in the joint. In many cases the injured synovial membrane undergoes hyperplasia resulting in hypertrophied synovial fringes which give a history of repeated pinching. We believe that the latter condition is caused much more frequently by the irritation of a hypermobile or torn cartilage, fractured tibial spine, loose body or the recurrent trauma due to an instable joint. Later operation on injured knees often shows multiple lesions all of which are capable of producing symptoms. Frequently only one structure is damaged by the acute trauma and the others are secondary to the abnormal joint mechanism resulting from it.

One must bear in mind that in consideration of acute injuries of any joint preexisting pathological processes made be present which often confuse the clinical picture. The condition most frequently met with in the middle age or older group is hypertrophic arthritis. In this condition the ligaments are less pliable and the amount of force required to cause the ligamentous injury is correspondingly less. It has been our observation that joints so affected are much slower in recovery. We do not believe that single acute injury can cause the hypertrophic arthritic changes but certainly these joints are much more susceptible to injury and are slower in clearing up.

Fractures about the knee joint do not come within the scope of this discussion but it must be borne in mind that most of these cases are accompanied by damage to the ligamentous structures. The recognition of this fact and the proper care of the injured ligaments is just as important as the care of the fractured bone. Unless this is fully appreciated many of our cases of fractured knees will have a perfect anatomical result so far as the bone is concerned but still be disabled on account of the unrecognized and untreated damage to the soft tissues.

In conclusion, we would again emphasize that in acute injuries of the knee regardless of which structures are most seriously

damaged the most important points of adequate treatment are the avoidance of prolonged distention of the capsule, protection of the damaged structures and maintainance of muscular tone of the thigh muscles.

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POST-TRAUMATIC INFECTIONS OF THE EXTREMITIES*

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THE extent of infection developing after trauma to the extremities depends upon the nature of the wound and the organisms with which it is contaminated. Traumatic lesions may be classified as abrasions, incised, punctured, lacerated, contused and gunshot wounds. The organisms usually found in such wounds include the staphylococcus, streptococcus and the anaerobes that produce tetanus and gas gangrene. For purposes of this discussion the subject will be approached from the standpoint of the usual types of infection encountered in these various wounds.

ABRASIONS

Since these are superficial lesions destroying the epidermis and opening a portal in the deeper layers of the skin and lymphatics, only the aerobic organisms capable of developing in and immediately under the skin are likely to set up infection. If the staphylococcus gains a foothold, characteristically there will be small pustules developing under crusts that form over the abraded area with a narrow red zone of inflammation in the skin immediately adjacent to the abrasion. If not aggravated by further trauma, such lesions tend to remain localized and superficial causing few symptoms. If the streptococcus predominates, the infection progresses more rapidly, redness about the margin of the injured part appears characteristically and spreads quickly for a considerable distance. Lymphangitis and lymphadenitis in the regional lymph-nodes will probably develop within a few hours and there will be increased heat, redness and intense pain associated with some fever. Blisters may appear on the surface and at the skin margins. The secretion from this type of wound is usually thin, watery and slightly blood tinged especially in the early stages. The diagnosis is based upon the clinical picture together with smears and cultures from the secretions. The infection usually subsides without invasion of the deeper tissues and most of the acute symptoms disappear if the resistance of the patient is good and proper measures of treatment are instituted early. Because such wounds do not form a suitable medium for the development of tetanus bacilli, this disease is seldom seen but has been known to occur following simple abrasions.

INCISED WOUNDS

A clean-cut wound made with a sharp instrument produces a minimal amount of tissue injury. Even when numerous organisms are introduced into such wounds, the resultant infection may be slight because the defensive forces of the body are not hampered by the presence of dead and dying tissue. Although it is possible for anaerobic conditions to exist in an incised wound when it penetrates to some depth, these organisms seldom propagate unless muscle tissue has been greatly damaged. The commonest invader, therefore, is the staphylococcus which produces a localized superficial abscess characterized by slight swelling, tenderness and a small surrounding area of redness in the center of which there develops a small focus of necrosis with white purulent exudate. When the staphylococci propagate in the deeper tissues, the infection is likely to be more extensive with greater swelling, tenderness, some fever and leukocytosis. As a rule, early localization and rapid healing follow evacuation of the abscess. A pure strepto-

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coccic infection seldom occurs in this type of wound but mixed infections of streptococcus and staphylococcus may develop to produce a cellulitis characterized by pain, swelling and diffuse tenderness with redness and brawny induration for a considerable distance about the wound. The symptoms appear rapidly and are associated with a greater systemic reaction of fever, malaise and leukocytosis. Localization and resolution of the process is sometimes slow. The discharge may be serous or seropurulent. Smears and cultures of the secretions will reveal a mixed type of infection with streptococci predominating.

PUNCTURED WOUNDS

A sharp or relatively sharp instrument may penetrate only the subcutaneous tissues or it may pierce the fascia and injure underlying muscle. The penetrating instrument is likely to carry infection from the skin surface; and if a foreign body such as a splinter or fragment of steel is left implanted in the deeper tissues, the infection is likely to be much more serious. Because there is more tissue damage and anaerobic conditions prevail, there is more danger of the development of infection than in the incised wound. The staphylococcic infection, evidenced by the slow development of a localized abscess in the subcutaneous tissues, is the commonest sequel. Throbbing pain, followed by diffuse swelling and tenderness, and perhaps some febrile reaction will persist for two or three days during which time there will be little or no discharge. Frank fluctuation appears if tissue necrosis and pus formation take place and when the abscess is incised and evacuated, the acute symptoms disappear.

When a punctured wound penetrates fascia and muscle, anaerobic conditions favorable to the development of tetanus and even gas gangrene prevail in the deeper parts of the tract. The development of tetanus may cause little inflammatory reaction unless other organisms are propagating at the same time. Although the wound may heal superficially, in the deeper parts a small granulating focus may be present which causes active tetanus. The early clinical picture is, therefore, dependent upon the activity of mixed infection rather than tetanus alone. Consequently, the symptoms and physical signs will be those of a staphylococcus or less frequently gas gangrene. The only positive meant of demonstrating the presence of tetanus organisms is by culture taken from the depths of the wound or better still from the débrided muscle.

The symptoms of tetanus are caused by exotoxins which are produced rapidly and absorbed by the lymphatic vessels. These enter the general circulation, are carried to the neuromuscular end plates and then ascend the motor nerves of the spinal cord and brain to become inseparably fixed in the nerve tissue. The rate of ascension of toxin in the peripheral motor nerves is comparatively rapid. Diffusion up and down the spinal cord produces a muscular spasm resulting in convulsions.

The symptoms of tetanus depend upon the virulence and number of organisms and the resistance of the individual. The first symptom usually is pain in the region of the muscles of mastication followed by spasm of the masseters. Once trismus has begun, it is progressive and results in the characteristic picture of "risus sardonicus." The jaws are tightly fixed and the patient is unable to swallow even fluids. Muscles of the neck and back then become involved, and as they go into tonic spasm, produce the typical appearance of opisthotonos. The abdominal muscles subsequently become rigid and the normal abdominal excursions of the diaphragm are inhibited, thus diminishing vital capacity and producing varying degrees of cyanosis. Convulsive seizures occur as a result of very slight stimulation and vary in duration and intensity. Occasionally, they are so violent that fractures of the spine or other bones may occur. Subsequently, muscles of the extremities become affected, particularly the flexor group. The temperature may be slight or great and is apparently of no

prognostic importance. Profuse perspiration and profound exhaustion follow the repeated spasms and death by asphyxiation may occur from a prolonged spasm involving the diaphragm and laryngeal muscles.

If the pathogenic anaerobic organisms (Clostridium welchii, Clostridium œdematiens or Clostridium œdematis-maligni (vibrion septique)) are introduced into traumatized muscle and especially if circulation in the part is diminished from swelling or constriction, typical signs and symptoms of gas gangrene may develop rapidly. These will be discussed under gunshot wounds. The diagnosis is based upon the history of a deeply penetrating wound through soiled clothing and skin which has not been treated by suitable preventive measures such as débridement and early implantation of sulfathiazole and the rapid development of the clinical picture of gas gangrene.

LACERATED WOUNDS

Lacerated wounds, whether superficial involving subcutaneous tissue or deep involving the fascia and muscles, contain a large amount of damaged tissue favorable to the growth of any of the contaminating organisms. In a superficial wound, however, the anaerobes have less opportunity to gain a foothold so that the accompanying infection is likely to be produced by the staphylococcus or a mixture of staphylococcus and streptococcus. When the staphylococcus alone is responsible for the infection, the symptoms develop slowly, at first being marked by the swelling and redness incident to wound repair. As the infection progresses, these symptoms become more diffuse and intense and the discharge more abundant and of a seropurulent type. If the wound is closed, severe pain develops as swelling increases. There is less pain and swelling when the wound remains open. Rise in temperature and leukocytosis will also vary depending upon whether or not the wound is closed, being greater when it is. In the open wound infection will usually progress to involve all parts that have been lacerated but not beyond this point. Gradual subsidence usually follows when adequate drainage has been established. Staphylococci may be recovered from the wound secretions in large numbers after the first twenty-four hours. When the streptococcus or the streptococcus with the staphylococcus are the invading organisms, there may be rapid increase in swelling for a considerable distance beyond the wound margin with an outpouring of serosanguineous discharge, rapid rise in temperature, severe pain and leukocytosis. These symptoms are all aggravated in a closed wound and less severe and rapid in development in the open wound.

When the deeper structures have been lacerated, pure staphylococcic or the mixed staphylococcic and streptococcic infections may develop with symptoms similar to those described in the superficial wound except that the systemic reaction of fever and leukocytosis is greater. The wound is deep, the muscle tissue damaged and anaerobic conditions prevail. For these reasons, the tetanus and gas gangrene organisms may propagate and produce a more serious type of infection. The clinical picture is one of gas gangrene appearing in the course of the first twenty-four to forty-eight hours and overshadowing such evidence of staphylococcic or streptococcic infection as may be present. This clinical picture will be discussed under gunshot wounds. The tetanus organisms, even though they may propagate along with the Clostridium welchii, will not produce symptoms until a week or ten days later.

CONTUSED WOUNDS

Contused wounds differ from the lacerated wounds only in the fact that the surrounding tissues are traumatized and there is frequently considerable devitalized muscle. A hematoma often develops and is easily infected producing a large abscess. The characteristics of infection developing in such wounds are not different from those described except that the extent of necrosis

and sloughing in the later stages is likely to be much greater.

GUNSHOT WOUNDS

Gunshot wounds are the severest of all because they represent a combination of punctured, lacerated and contused wounds with the addition of a foreign body. The character and extent of infection naturally vary with the type of projectile and the portion of the extremity traversed. It is usually true that the larger, blunter and slower traveling projectiles are the most dangerous from the standpoint of infection. Steel-jacketed bullets of high velocity may pierce an extremity even grazing or passing through the bone without introducing infection of any consequence. A ragged shell fragment of low velocity, barely penetrating muscular layers, is likely to produce the most serious infection. If the bones are shattered and spicules are driven into surrounding tissues, the possibility of infection proportionately increases. Therefore, compound fractures caused by projectiles other than those of high velocity, are most likely to be accompanied by severe infection. Wounds resulting from shotguns fired at close range in which massive loads of small or large shot penetrate muscular layers accompanied by bits of clothing, dirt and gun wadding will probably produce gas gangrene even when the bones are not shattered.

The deep implantation of various organisms with extensive damage to the surrounding tissues, the presence of foreign bodies and prevailing anaerobic conditions all combine to favor the development of gas gangrene and tetanus rather than a staphylococcic or streptococcic infection. Under such circumstances Clostridium welchii propagate rapidly and invade the muscles. Large amounts of toxin and gas are thus produced. These toxins cause death of surrounding tissue and intense edema and when absorbed, strongly affect the pulse rate, temperature and nervous condition of the patient. The gas, accumulating in the muscle layers beneath inelastic fascia, expands along intermuscular planes under considerable pressure, thus depriving the muscle of its blood supply. This causes additional necrosis and creates new culture media for the rapidly propagating organisms. Thus is initiated a vicious cycle which, when unbroken, results in rapid spread of the infection through all the muscles in the wounded extremity distal to the joint.

The presence of gas gangrene should be suspected from the nature of the injury and the history of failure to institute appropriate preventive measures. The outstanding symptom in the early stage twelve to twenty-four hours after injury is intense pain for a considerable distance around the wound. Although there is always some elevation of temperature during this same period, the characteristic finding is a rapidly rising pulse of full volume and of greater rapidity than would ordinarily accompany the moderate amount of fever. Restlessness, anxiety and thirst are typical. The dressings are frequently soaked with large quantities of serosanguineous discharge characteristically brick-red in color and with a distinct, pungent, nauseating odor. Upon removal of the dressing, the entire circumference of the limb for some distance above the wound is often greatly swollen and tense and the skin is shiny, perhaps mottled, copper-colored and covered with blebs and blisters. Pressure over any part of the distended limb produces intense pain and as the fingers are pushed toward the wound, additional serosanguineous fluid may be expressed, which when carefully observed, is found to contain small gas bubbles. Sometimes these bubbles cannot be seen unless a forceps is inserted into the depth of the wound and sufficient pressure is used to express fluid from the muscular layers. Roentgenograms made at twenty-four to forty-eight hours after injury will reveal the presence of gas bubbles in the muscular tissue for some distance about the wound. The white blood count is usually high but the red blood cells rapidly decrease as

shown in counts made at intervals of four hours. Smears from the depth of the wound which show large numbers of short Gram-positive nonmotile rods and very few other organisms, although not proving the diagnosis, are helpful when considered with other characteristic findings.

TREATMENT

The only effective treatment of post-traumatic infections is preventive. Early and thorough débridement of punctured, gunshot and lacerated wounds has long proved valuable. Unless the débridement is begun within six hours of injury and is thorough, experience has shown that it is unsafe to close such wounds primarily. Staphylococcic infections, gas gangrene and tetanus have developed following such early débridement and closure even when skillfully performed. For these reasons, tetanus antitoxin and the combined tetanus and polyvalent gas gangrene antitoxin should be administered routinely. However, primary healing without complications cannot be confidently anticipated by these measures alone. From 25 to 50 per cent of the wounds will break down, suppurate and heal by secondary intention. Such a course is particularly undesirable in compound fractures. The introduction of active immunization against tetanus by the use of tetanus toxoid has proved to be an effective prophylactic measure. A second immunizing dose of the toxoid should be administered shortly after a wound is incurred even when the patient has recently received a full immunizing course of tetanus toxoid.

Since the advent of the sulfonamides, it has been found that their implantation in the débrided wound together with oral or intravenous administration of the drug following the operation has a definite inhibitory action upon the development of infection of all types. Clinical reports indicate diminished incidence but not absolute control of wound infections when these drugs are implanted at the time of débridement performed six hours after injury. Experimental work has shown that *immediate implantation* of the sulfonamide derivatives at the time of the accident followed by early débridement and re-implantation of the drug is most effective in preventing all infections. It appears from the experimental and clinical evidence that the use of sulfathiazole as a first aid dressing is essential in the prevention of infection of post-traumatic wounds. However, the immediate implantation of sulfathiazole in infected wounds cannot be relied upon as the sole means of preventing infection. Débridement within six hours followed by re-implantation of sulfathiazole may be expected to inhibit the development of infection with such certainty that the wounds may be closed and primary healing expected. These measures, properly employed, are such a valuable adjunct that even internal fixation of fractures may be used with reasonable safety.

Based upon these experimental and clinical observations, the ideal plan of treatment for severe punctured, lacerated and gunshot wounds includes: (1) immediate implantation of 5 to 20 Gm. of sulfathiazole in the wound covered with an occlusive dressing; (2) the use of transportation splints and appropriate shock treatment; (3) thorough débridement within six hours of injury; (4) re-implantation of 5 to 15 Gm. of sulfathiazole depending upon the size of the wound and the extent of contamination; (5) primary closure of the wound, and, in the case of compound fractures, splinting such as would be used for simple fractures; (6) intravenous or oral administration of sulfathiazole postoperatively for five to seven days controlled by the blood level; and (7) daily blood counts, urine examinations and blood level determinations for control of toxic effects of the drug.

SUMMARY

The extent of the development of post-traumatic infections of the extremities depends upon the nature of the wound and

the type of organisms introduced. These injuries may be classified as abrasions, incised, punctured, lacerated, contused and gunshot wounds. A staphylococcic or streptococcic infection may develop following abrasions but these are ordinarily of little consequence. The development of infection after incised, punctured or lacerated wounds causes little systemic reaction if the lesion is superficial. However, in deep wounds in which the fascia and muscles have been penetrated, the introduction of streptococcus, staphylococcus and anaerobic organisms is productive of more acute symptoms. The most serious type of infection following a deep puncture is tetanus, characterized by pain and spasm of the masseters, trismus, opisthotonos, convulsions and death. Tetanus antitoxin and combined tetanus and polyvalent gas gangrene antitoxin should be administered routinely to all patients with incised, lacerated or punctured wounds. The severest type of infection follows gunshot wounds because they represent a combination of punctured, lacerated and contused wounds with the addition of a foreign body. Under anaerobic conditions, the deep implantation of various organisms, extensive tissue damage and the presence of foreign bodies encourage the development of gas gangrene. This picture is characterized by intense pain for some distance about the wound, rapid pulse rate out of proportion to the moderate temperature, brick-red serosanguineous discharge and distinct, pungent, nauseating odor. The prophylactic measures of immediate implantation of sulfathiazole as a first aid measure, early débridement and reimplantation of sulfathiazole will prevent the development of this dreaded disease.

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Lesions of the Gastrointestinal Tract

CHRONIC ULCERATIVE LESIONS OF THE MOUTH*

INCIDENCE OF CANCER IN 2,077 CASES AT BARNARD HOSPITAL

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THREE out of every four chronic ulcers of the mouth are malignant. Only one out of four is benign. Such is the crux of a study of 2,077 mouth lesions used as a basis for this paper. This represents a review of every mouth ulcer from 1904 to 1941 seen at the Barnard Free Skin and Cancer Hospital or Clinic, provided only that microscopic study was made of the lesion.

The significance of this observation, that the majority of chronic mouth ulcers are malignant, cannot be stressed too strongly. It means, to the practitioner or dentist, that any ulcer of the mouth which fails to heal within a week or two is probably due to cancer. Were this fact widely appreciated, the mortality from mouth cancer could be markedly reduced. For early cancer of the mouth is generally curable if properly treated. Proper treatment depends, in large part, upon proper diagnosis.

While the practitioner or dentist is listening to the history of a patient stating that he has had an ulcer in his mouth for a week or more, he may assume at once that the chances are, some three to one, that this ulcer is malignant. He will mentally indict the lesion as malignant before he even looks at it, and his attitude will be, throughout examination, to assume it to be malignant unless positively proved benign. He will not be easily misled by the patient's idea that the sore in his mouth is caused by a bad tooth, or a poor denture, or that the lip has been burnt by a cigarette or by a pipestem. He will not lull himself into false ease by the knowledge he may already have that his friend, the patient, is syphilitic or tubercular.

What he finds when he examines the patient, his mouth and the ulcer, this paper will attempt briefly to describe. Besides 2,077 pathologically proved mouth ulcers of the Barnard Hospital series, reference will be made to 406 mouth cancers from the private patients of the late Dr. Ellis Fischel;[1] and also to 187 lip cancers from the private patients of Dr. Fischel and of the writer. An attempt will be made to give relatively more attention to the commoner cancerous ulcers of the mouth, and less to the rarer noncancerous chronic mouth ulcers.

SQUAMOUS CELL CARCINOMA OF THE MOUTH

Of the 2,077 mouth lesions of the Barnard Hospital series, 1,561 were malignant, an incidence of 75.2 per cent. Of these, twenty-eight were sarcoma, eight adenocarcinoma, two basal cell, and the remaining 1,523 were carcinoma of the squamous cell type. The diagnostic features of these 1,523 squamous carcinomatous ulcers of the mouth were as follows:

Sex. Squamous carcinoma of the mouth was found to be chiefly a disease of men, and 91.1 per cent of Dr. Fischel's 406 patients with the disease were males. This

* From the Department of Surgery, Washington University Medical School and Barnard Free Skin and Cancer Hospital, St. Louis.

percentage is given by other authorities anywhere from about 60 per cent to over 90 per cent.[2]

Lip cancer was found almost exclusively in men, and all of Dr. Fischel's 124 private patients* with squamous carcinoma of the lip were males. Lip cancer, however, did occur in three women of the Barnard Hospital series, leaving a balance of 784 men with squamous lip cancer. These three women comprised two elderly and inveterate pipe smokers, and one younger woman, also a smoker.

The lip cancer of women is generally not squamous cell but basal cell in type. It begins in the skin of the upper lip, and if untreated may eventually ulcerate into the mouth, as it did in two instances at Barnard.

Tongue cancer occurred relatively more often in women, and of Dr. Fischel's 166 patients, 10.7 per cent were females. Carcinoma of the buccal mucosa occurred even more frequently in women, and Dr. Fischel's 106 patients comprised 14.7 per cent females.

Age. The majority of patients of this series were sixty years or over in age, and mouth cancer was frequently observed in all the later decades of life. Very many patients between fifty and sixty also had mouth cancer, but below fifty the disease became increasingly rare. The youngest was a boy of nineteen with squamous cell carcinoma of the lower lip; Hall and Bagby in 1938 recorded fourteen other examples from Barnard Hospital of proved mouth cancer in the second and third decades of life.[3]

Complexion. Mouth cancers, like skin cancers, were more common in fair-haired, thin-skinned, freckled individuals; rarer in black-haired, thick skinned patients, and very uncommon in the negro race.

Habitus. The lower lip was notably prominent in some patients developing cancer of the lip.

* The other group of 187 lip cancers, referred to in the article, consists of these 124 private patients of Dr. Fischel's, supplemented by twenty-three other private patients.

Associated Conditions. Cancer was very rare in a clean mouth. Cancer was usually found to be associated with foul teeth or

Fig. 1. Typical carcinoma of the right margin of a leukoplakic tongue. Note the rolled, raised border. Diagnosis verified microscopically.

infected gums, or was seen in the mouths of edentulous old men who chewed or smoked a great deal.

Leukoplakia commonly accompanied carcinoma of the buccal mucosa, and was prone to occur within the cheek on the side where the quid of chewing tobacco was held. Leukoplakia accompanied carcinoma of the tongue somewhat less often than it accompanied carcinoma of the buccal mucosa. Leukoplakia also was fairly common with carcinoma of the lip. In the lip, a white patch was prone to occur in the particular spot where a hot pipestem was habitually smoked, and carcinoma was apt to arise later in this location.

The tongue of old men with mouth cancer at times showed evidence of vitamin B deficiency and other nonsyphilitic signs. The tongue was sometimes smooth, with papillary atrophy of varying degree, or deep fissures or cracks and irregular borders. The tongue at other times was

snowed under a mantle of leukoplakia, or showed papillary hyperplasia. Many tongues were heavily coated; some dark brown in color. The breath was often foul.

Signs of syphilis also occurred in some tongues, with scarring and atrophy. Syphilis in this series more commonly accompanied cancer of the tongue than cancer of the lip or buccal mucosa. It is well worth remembering, however, that cancer can exist with syphilis and that a diagnosis of syphilis does not exclude cancer. Fatal mistakes have been made in this connection.

Chronic irritation was observed in many mouths and examples of cancer arising near a jagged tooth were not wanting. Many of the mouths were unusually foul, with snags, exposed roots, pyorrhea, dead teeth, or root abscesses quite the rule. Irritation from improper dentures or poor bridgework was sometimes seen. Occasionally, however, cancer was seen in a really clean mouth, as for example a very early carcinomatous ulcer seen by the writer between the well kept upper lateral incisor and canine tooth of a young woman of twenty-eight who smoked much and had the habit of brushing the place very hard with a toothbrush. This was unusual, however, and far commoner was it to see cancer in the mouth of a blonde or red-haired, freckle-faced neglectful farmer, tobacco-chewing, pipe-smoking, dirty-toothed or edentulous.

Most of the men and many of the women with cancer of the mouth used tobacco. Yet cancer did occur at times in the mouth of an individual who denied ever having used tobacco in any form. This study in no way attempts to prove whether tobacco influences the origin or growth of cancer in the mouth.

Previous History. Many patients had had tooth extractions, often in the presence of a cancer unrecognized by the party removing the teeth. Mistaking cancer of the mouth for an infection, an ulcer, or a "gum-boil" or sore of some kind was quite common; the result was treatment by incision, burning, paste, electricity, etc., often quite unwarranted. A history of a sore mouth or a sore tongue often preceded the actual development of a mouth cancer.

Symptoms. (*a*) *Lip.* The symptoms of 187 private patients with cancer of the lip were studied especially carefully. The time elapsing between the occurrence of the first symptom and consultation was on the average twelve months. The shortest time was two weeks and the longest was a matter of years. A dozen patients reported five years or more after observing a lesion, probably noncancerous at first, on the lip. Recently, patients have been reporting sooner. Previous to 1930, the average time was one year and five months; since 1930, it has been ten months.

The lesion was described by seventy-three of the 187 patients as a sore or ulcer, and as a fever blister by thirty. It was called a growth, nodule, tumor or lesion by twenty-one, a white spot or leukoplakia by seventeen, a wart, corn, or horn by eleven; six called it cancer, six a pimple, and four a brown spot.

Twenty-one patients complained of no pain; twelve had pain. Itching, dryness, pulling, or smarting was felt by seven. One said his blister enlarged when the moon was full. Some patients complained of tenderness or thickness of the lip, or thought it rough or chapped. Three had a sore or rough mouth.

Progress of the lesion was described by eighty-nine as slow growth, or failure to heal, or healing then splitting. Only six bled. The cancer was attributed by eighteen patients to razor cuts, blows, dental plates, extractions, smoking, burns or bites. A dozen or so noticed lumps or knots under the jaw, two considering them boils, two a pain in the neck.

(*b*) *Tongue and Buccal Mucosa.* The duration of symptoms at the time of first examination was less with intra-oral carcinoma than with carcinoma of the lip, and was generally less than six months. This led to the deduction that carcinoma within the oral cavity was more bothersome to the patient, and generally more rapid in rate of growth than was carcinoma of the lip. Tongue cancers grew especially rapidly,

less than 6 per cent of the patients surviving over a year untreated.

In general, more patients with intra-oral cancer complained of pain than complained simply of tumor. Thus, many said they had a sore in the mouth or an ulcer or a sore mouth, while relatively fewer stated they felt a mass, lump, tumor or rough spot. Although difficult to evaluate, a certain amount of pain seemed of rather frequent occurrence, though the pain was rarely marked in degree. In this respect intra-oral cancer seemed to differ from lip cancer.

As with lip cancer, so also did patients with intra-oral cancer frequently blame their "sore" on a tooth snag or their white spot to tobacco. Rather often patients thought they had burned their tongues or had a raw tongue from smoking; some said they had painful or stiff tongues; others had difficulty in eating. A few thought they had a "gum-boil" and others a plate ulcer. Few complained of bleeding. There was occasional hoarseness, and sometimes knots, kernels, lumps, or tumors were noted in the neck.

Physical Findings. (*a*) *Lip.* Squamous cell carcinoma commonly occurred in the lower lip, and 120 of Dr. Fischel's 124 cases were located in the lower lip, only four arising in the upper lip. Basal cell carcinoma arose more often in the upper lip, particularly in women. Basal cell carcinoma arises from the skin proper, squamous cell carcinoma from the vermilion or mucous surfaces of the lip. Basal cell carcinoma rarely extends into the buccal cavity itself when properly treated, hence only two examples of basal carcinoma have been recorded in this series, both at Barnard Hospital.

The average diameter of the squamous carcinomas recently observed was 15 mm. The diameter was larger in the earlier cases. The smallest lesion was 5 by 3 mm. and the largest involved the entire lower lip.

Characteristic of squamous carcinoma of the lip was an ulcer or a button-like lesion with rolled, pearly borders, often with induration and infiltration into the deeper tissues. A scale or crust or yellow membrane covered the granular raw base of some of the lesions. Leukoplakia of the lip or buccal surfaces was frequently noted, and atrophy or scarring of the mucous membranes sometimes occurred. Some of the lesions were ulcerative, some were papillary, and many were button-like; some resembled a wart, some were nodular. Differentiation, clinical or pathological, between early cancer and hyperkeratosis or leukoplakia was sometimes difficult.

Advanced, untreated carcinoma extended onto the alveolus and the buccal mucosa, destroyed the lip, and even involved periosteum or bone. The advancing cancerous edge was usually nodular, hard, with a tendency to ulcerate, leaving a foul, sloughing, bleeding mass behind. Drooling and malnutrition completed the unattractive picture of the very advanced group, but it was interesting how extensive the involvement could be in the lips of some patients with relative freedom of the cervical lymph-nodes from metastases. The converse was sometimes observed: a small primary lesion with extensive cervical metastases.

(*b*) *Tongue and Buccal Mucosa.* The average size of the tongue lesions when first seen has recently been 3 cm., and for lesions in the buccal mucosa, 4 cm. Lesions 2 cm. or under were relatively rare.

Extensive lesions of the tongue were common, and it was all too frequent to see much of the organ involved. Lesions usually arose on the dorsum of the tongue, but some occurred beneath it spreading onto the floor of the mouth and the inferior alveolus. Lesions of the middle third of the tongue were apt to spread onto the pillars and palate. Lesions of the posterior third spread onto the pharyngeal wall, tonsil, and pyriform fossa.

Extensive lesions of the buccal mucosa were common. They spread to the alveolus, either inferior or superior, or hard palate, and frequently involved the fornix or the angle of the mouth.

Carcinoma of the tongue or buccal mucosa in general resembled a dirty ulcer,

usually with raised, hard borders, often with a granular base, bleeding easily, and apt to be covered with a dirty, yellow membrane. The raised, rolled border usually was characteristic; it was rarely pearly in appearance like it was on the lip. The indurated feel of the border often conveyed a better sense of carcinoma than did the look of the rolled edges.

Carcinomas were of two general classes: ulcerative or papillary. The ulcerative lesions burrowed into underlying tissue, sometimes down to bone. The base almost always felt hard and tended to draw or pucker tissues to it. Thus a carcinoma of the right side of the tongue, middle third, tended to draw to it the velum and floor of the mouth, and to cause deviation to the right of the protruded tongue; or a carcinoma of the fornix would invade the pterytoid fossa and limit the bite as a result. Some carcinomatous ulcers of the buccal mucosa penetrated the cheek, occasionally completely perforating it. Papillary carcinomas were cauliflower-like, ulcerating growths with the same characteristics except for a slower rate of growth.

Neck. The cervical lymph-nodes are of great importance with cancer of the mouth. These nodes were found to be palpable in a majority of cases. Often they were small and soft and obviously contained no metastases, an observation usually confirmed when a neck dissection was subsequently performed. In another group were nodes which were somewhat hard, shotty, suspicious of cancer, yet not enlarged. This suspicion of cancer, in such instances, was later verified microscopically in almost 50 per cent of the operative cases. Then came nodes enlarged, hard, still movable, and obviously carcinomatous, an observation generally confirmed in the operative cases. Finally were the group of nodes hard, fixed, and sometimes ulcerating through the skin, the group of hopeless cases.

Cancer spread from the lip first to the submental, submaxillary, buccal, or anterior parotid group of lymph-nodes, homolateral, contralateral, or bilateral, some times years after the cure of the primary tumor. From the buccal mucosa, cancer spread first usually to the above group of nodes, homolateral always, at a rate generally faster than from the lip. From the tongue, carcinoma usually spreads to the jugulodigastric lymph-node, and almost as quickly to the remaining nodes of the deep jugular chain, extension being generally homolateral, but often contralateral or bilateral, and at much the most rapid rate of the group of mouth cases.

Beyond these first lines of defense, metastatic carcinoma spread, in the absence of neck dissection, to the deep jugular chain of nodes, at first above, then below the level of the tendon of the omohyoid muscle. Finally, metastases spread to the posterior triangle of the neck, and, very rarely, below the level of the clavicle either to mediastinum or elsewhere.

Cancer, in the individual node, entered the peripheral lymph channels, formed follicular deposits, then filled all the node, finally penetrated the capsule, causing the node to become fixed to the carotid artery, mandible, vertebra, or skin, acccording to varying circumstances.

Biopsy. Biopsy was often used to confirm the diagnosis of cancer in the mouth, occasionally in the neck. Gross, not punch biopsy, was favored. With or without infiltration with a ½ per cent solution of procaine hydrochloride with adrenalin four drops to the ounce, a small piece was accurately cut with a sharp knife from the rolled border of the tumor, including a section through the base of the ulcer and from the adjacent normal mucosa. Light cautery usually effected hemostasis when necessary. Sutures for hemostasis were not employed. The tissue, fixed in 10 per cent formalin, was embedded in paraffin, stained generally with hematoxylin and eosin, and examined by the pathologist at leisure. Frozen sections were rarely used, being much less satisfactory, and not worth the time so saved.

An index of diagnostic acumen was afforded by a study of the 187 lip lesions. The clinical diagnosis was proved correct in 172 of the 187 cases, or 92 per cent. Of

the fifteen lesions diagnosed incorrectly, ten lesions diagnosed clinically malignant proved pathologically benign, and five diagnosed clinically benign proved pathologically malignant.

Biopsy itself was not perfect. A number of lesions, clinically carcinomatous were reported pathologically benign, and later proved actually malignant. Thus a cancer in the lip of a patient previously treated with radium, appeared clinically still to be malignant, but biopsy was reported benign. Nevertheless, because of the clinical conviction that some cancer remained, a "V" excision of the lip was performed, and the clinical diagnosis vindicated by the final pathological section which showed squamous carcinoma. The better trained surgeons looked at the microscopic slides, and correlated the pathological with the clinical aspects of the case.

The most difficult differential diagnosis was between radiation ulcer of the mouth and cancer. Biopsy was almost always necessary to settle this question.

Regional Incidence of Cancer in the Mouth. The 1,523 squamous cell carcinomas of the mouth at Barnard Hospital occurred 787 in the lip, 564 in the buccal mucosa, and 172 in the tongue. For the lip, the percentage was 51.7, for the buccal mucosa, 37 per cent; for the tongue 11.3 per cent. In Dr. Fischel's 406 carcinomas, for the lip the percentage was 30.5; for the buccal mucosa, 28.6 per cent; and for the tongue 40.9 per cent. At Barnard Hospital there appeared to be relatively more cancer of the lip and relatively less cancer of the tongue than among Dr. Fischel's private patients.

Partially accounting for these disproportions among tongue cancers were (*a*) the fact that in the Barnard series pharynx was not classed under tongue, whereas in Dr. Fischel's series it was; and (*b*) that the Barnard series, being based solely on microscopic data, of necessity omitted the many advanced tongue cancers on which no biopsy was performed. Accounting for the disproportion of lip cancers might be the fact that the indigent farmers at Barnard were, in general, more exposed to the Missouri sun than were the presumably more protected private patients of Dr. Fischel's series.

Generally, and for convenience, carcinoma of the buccal mucosa has been made to comprise carcinoma of the alveolus, palate or velum. Carcinoma of the tongue has comprised carcinoma of the floor of the mouth, fauces, and, in Dr. Fischel's series, also tonsil, pharynx, and pyriform fossa.

OTHER MALIGNANT MOUTH NEOPLASMS

Of the 2,077 mouth lesions at Barnard, 1,561 were malignant neoplasms. Of these 1,561, 1,523 were squamous cell carcinoma, and these have been described. There remain thirty-eight malignant neoplasms, two of which were basal cell carcinoma of the upper lip of females, also referred to above. The balance, thirty-six cases, comprises twenty-eight sarcomas and eight adenocarcinomas.

(*a*) *Sarcoma.* The twenty-eight sarcomas comprised twelve giant cell sarcoma of the jaw or gum, seven spindle cell sarcoma of the jaw or mouth, three small round cell sarcoma of the jaw, two fibrosarcoma of the lip, and one each of angiosarcoma of the lip, lymphosarcoma of the jaw, osteogenic sarcoma of the gum, and sarcoid of the mouth.

Sarcoma sometimes attained huge proportions with distortion in the soft parts from which they arose. They involved the mouth secondarily. Some were slow, some rapidly growing, and a few resembled epulis. They tended to occur in children and young adults, as for instance a giant cell sarcoma of the gum of an 8 year old boy and a spindle cell sarcoma of the cheek of an eleven-year old girl. In sex, as in age incidence they differed from carcinoma. Some were hard, others soft, but since ulceration in the mouth was of secondary importance they need not further be discussed here.

(*b*) *Adenocarcinoma.* The eight adenocarcinoma arose four in the buccal mucosa, two in the palate, one in the tongue, and one in the lip. They were characterized by

indolent ulcers, slow-growing, with a less marked elevated, rolled, indurated border than squamous carcinoma. Biopsy was necessary for diagnosis.

BENIGN MOUTH NEOPLASMS

Of the 2,077 mouth lesions at Barnard, 180 were benign, 1,561 malignant. The malignant neoplasms have been described. The majority of the benign neoplasms comprised sixty-three epulis and fibroma, thirty-eight angiomas of various types, thirty-one polyps or papillomas, and twenty-six cysts.

(*a*) *Epulis and Fibroma.* There were forty-one examples of epulis, twenty-two of fibroma. Epulis was generally a tumor of late childhood, young adult life, or middle age, and was fairly rare in males. In this series, the forty-one examples all involved the gums primarily, and sometimes the adjacent structures secondarily. A characteristic history was of a persistently growing mass, sometimes of a year's duration, painless, usually unsightly because of its prominent location in the gums, rarely in a really clean mouth. The tumor was soft and elastic, or a hard mass between the front teeth, sometimes pedunculated or sessile. Both gums were apt to be hyperplastic. Extension was usually limited to root sockets and alveolus and the diagnosis rarely in doubt. Occasionally, a suspicious epulis proved to be sarcoma microscopically.

Fibroma in some respects resembled epulis. The distribution in the mouth, however, was different, and the twenty-two examples comprised ten of the tongue, nine of the buccal mucosa, including only four on the gums, and three on the lip. This tumor, relatively more often seen in males than was epulis, generally presented a slow-growing small sessile lump, usually soft. The chief differential diagnostic difficulties were to distinguish it from other benign tumors such as simple cysts.

(*b*) *Hemangioma and Lymphangioma.* There were thirty hemangiomas or angiomas, and eight lymphangiomas. Hemangiomas occurred sixteen times on the lips or adjacent mouth, and fourteen on the tongue. The tumors were soft, slow-growing over many years, painless, rarely bleeding, very rarely ulcerating, usually fairly small, characteristically blue and soft, occurring at almost any age.

Lymphangiomas occurred in five tongues, two lips and one gum. These were soft, slow-growing, bulky tumors, with indefinite borders, generally of younger years, and very rarely ulcerative.

(*c*) *Papillomas and Polyps.* There were twenty-nine papillomas and two polyps. The papillomas were distributed as follows: twelve on tongues, nine on lips, three on buccal mucosa and one on the gum. These lesions were slow-growing, localized, soft enlargements of a papilla, occasionally ulcerating, rarely painful, obviously benign, occurring in later years, with almost equal distribution between the sexes.

(*d*) *Cysts.* Of the twenty-six cysts, twenty-three were simple and three dermoid. The simple cysts occurred in eight lips, seven jaws, five buccal mucosas and three tongues. The dermoids occurred in one tongue and two cheeks. Cysts included ranula of the floor of the mouth. The age group was young, some cysts being congenital. Symptoms were due to the size of the cysts and their situation. Since cysts rarely ulcerated, no further description is necessary. Bone cysts and tumors have been omitted from this study.

(*e*) *Miscellaneous.* The balance of the benign tumors comprised nine mixed tumors, five on the palate, three on the buccal mucosa, one of the lip. These rarely ulcerated, and were slow-growing, small tumors of younger ages, sometimes diagnosed without biopsy. The balance of benign tumors comprised three adenomas and three myxomas of mucous surfaces, and three nevi of the lip.

BENIGN ULCERS OF THE MOUTH

There were 186 inflammatory ulcers among the 2,077 Barnard cases. Of the 186 cases, 149 comprised simple inflammatory

ulcers, and thirty-seven were granulomatous lesions including tuberculous and syphilitic lesions.

(*a*) *Simple Inflammatory Ulcers.* There were six subdivisions of the 149 ulcers in this category, namely, pyogenic ulcers, radiation ulcers, ulcer due to dental plates, syphilitic ulcers and tubercular ulcers, and ulcers associated with carcinoma.

Simple pyogenic ulcers of nonspecific nature formed about 65 per cent of the 149 ulcers of this group. These were of three general types: those occurring alone, those occurring with leukoplakia, and those occurring along with cancer, a type comprising some 25 per cent of the 149 ulcers. The borders were generally flat, soft, fairly regular. The base was usually granular and soft with little infiltration. In size they were small. Some were mere cracks or fissures. When they occurred with leukoplakia they were especially dangerous because of the likelihood that they contained cancer. The etiology of simple ulcers was not easy to establish when carcinoma, syphilis and tuberculosis had been excluded by general physical examination, and by appropriate chest roentgenograms and laboratory tests, including Kahn or Wassermann reactions, and by dark field examinations. Some were due to Vincent's disease; some were nonspecific.

Radiation ulcers formed about 10 per cent of the 149 ulcers. Radiation ulcers followed the treatment of some mouth cancers by radium, and these ulcers sometimes closely resembled the original cancerous ulcer. Radiation ulcers were apt to be indolent and very dirty. The borders were generally indurated but were less rolled than were the borders of a carcinomatous ulcer. The base of a radiation ulcer generally contained a foul slough due to infection with saphrophytic and anaerobic organisms in symbiosis with pyogenic bacteria. The base as a result was apt to be infiltrated, fixed and hard, and often the cause of the pain which was a prominent feature of these ulcers.

Pain and chronicity were two important points in differentiating them from cancer. Biopsy or excision often proved necessary for diagnosis. It was always imperative to determine the presence or absence of carcinoma in such ulcers.

Plate ulcers formed some 10 per cent of the 149 simple ulcers. They occurred usually in women and were caused by ill-fitting dentures. The ulcers were generally of long standing, often painful. They were generally single and arose in the gum where the plate rubbed. They were usually small, their edges raised but slightly, and were soft, with a clean base, granular but not fixed. The diagnosis was easy when healing quickly followed the leaving out of the denture. Occasionally, excision was necessary for cure or for biopsy. At least one patient developed cancer later at the site of a plate ulcer.

Some syphilitic ulcers, especially on the tongue, clinically and histologically appeared to be mere chronic pyogenic ulcers. Less than 10 per cent of the 149 ulcers of this group fell under this heading. These ulcers resembled the simple ulcers in the preceding paragraph, but they were more apt to be dirty and ragged, and to occur in young adults with positive serology.

Tuberculous ulcers formed about 5 per cent of the 149 ulcers of this group. These ulcers were not unlike simple ulcers except that they were sometimes dirty and punched-out, occasionally quite painful. Associated pulmonary tuberculosis was the rule in these cases.

(*b*) *Granulomatous Lesions.* There were thirty-seven granulomatous ulcers of which fourteen were tubercular, five syphilitic and the remainder of nonspecific origin. These tubercular and syphilitic ulcers were so diagnosed microscopically, those of the preceding section clinically without a characteristic histological picture.

Tuberculous lesions were fairly deep, dirty, punched-out ulcers, sometimes with rolled borders, occurring usually in patients with pulmonary tuberculosis. Some of these ulcers were quite painful. The biopsy was usually characteristic.

Syphilitic ulcers were more rare than one might expect from the literature. To the five cases diagnosed microscopically must be added a small number described in the preceding section. The five cases showing typical histology occurred in one lip, two tongues and one was a gumma of the palate. The lip lesions were generally indurated and button-like ulcers with a rather dirty base, the induration sometimes being suspicious of carcinoma. Ulcers on the tongue were dirty and irregular.

For a discussion of mycotic and yeast infections of the mouth, the reader is referred to an article by Dr. M. Moore of Barnard Hospital.[4]

DEGENERATIVE LESIONS OF THE MOUTH

There were 150 degenerative lesions among the 2,077 pathological lesions at Barnard Hospital. Degenerative lesions comprised sixty-two leukoplakias, fifty-nine keratoses or hyperkeratoses and twenty-nine hyperplasias. Leukoplakia has already been described in some detail in the section dealing with mouth cancer, with which it was frequently associated. The sixty-two leukoplakias here under discussion occurred without carcinoma at the biopsy site, biopsy usually being taken to rule out carcinoma. The sixty-two examples of leukoplakia were distributed as follows: thirty-four in the buccal mucosa, twenty-two in the lip and six in the tongue. This distribution was similar to the distribution of leukoplakia when associated with mouth cancer. Leukoplakia generally occurred in the same relative age and sex groups as did carcinoma, namely, it was relatively common in elderly tobacco-chewing, blonde males.

The duration of leukoplakia was an important point in its differentiation from carcinoma. Often leukoplakia was of many years' duration. Most patients characterized it as a white patch usually symptomless, sometimes rough or cracking or ulcerating. Physical examination revealed a white or pinkish, granular, veil-like deposit in the leukoplakic regions, usually confluent, sometimes patchy, usually slightly raised, often dry. Cracks, fissures, and ulcers occurred with it, varying from simple small ulcers to frank carcinoma.

The importance of leukoplakia lay in its relation to carcinoma. Carcinoma fairly often arose in a patch of leukoplakia, and it was at times extremely difficult clinically, or even microscopically, to distinguish between leukoplakia and carcinoma. Some leukoplakic patches on the dorsum of the tongue or on the buccal mucosa were white or pinkish, thick, velvety and deeply fissured. While no true rolled border might be evident and no real induration of the base present, it was especially important to suspect cancer and to take an immediate biopsy, being sure to cut very deep. Negative biopsies in such cases were also to be suspected unless the entire lesion had been excised with a minimum margin of 1 cm. of normal tissue all around.

Leukoplakic white patches of the lip at times merged imperceptibly into cancer. The border would pile up and tend to become pearly, the centers to become pitted, depressed and ulcerative, and finally frank cancer to develop.

Keratosis or hyperkeratosis occurred in fifty-four lips and five tongues. These lesions occurred usually in the type of individual apt to develop mouth cancer. The keratotic patch was apt to occur in the spot on the lower lip over which a hot pipe stem habitually was held. It was apt to occur at the place where paper from a cigarette would stick.

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CARDIOSPASM

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Definition. Cardiospasm, second only to carcinoma as a cause of dysphagia, is a disturbance in the nerve-muscle mechanism of the esophagus whereby the esophagogastric junction remains in a state of partial or complete contracture and produces epigastric pain, dysphagia and regurgitation of food. In association with the area of contraction at the cardia there usually is more or less dilatation of the esophagus above the point of obstruction.

Various terms have been employed to describe the condition, none of which has been universally accepted because of uncertainty of the cause and exact nature of the disease. Among those that have been suggested are diffuse dilatation of the esophagus without anatomic stenosis, hiatal esophagismus, phrenospasm, achalasia of the cardia, esophagospasm, ectasis of the esophagus, idiopathic dilatation of the esophagus and congenital dilatation of the esophagus.

Frequency. Cardiospasm is responsible for symptoms in approximately one-third of all patients who suffer from difficulty in swallowing. Several thousand cases have been reported in medical literature, and some authors have reported several hundred cases.

Age, Sex, and Racial Distribution. Cardiospasm may be observed at any age, but is most frequently seen in the third decade of life. In the white race males are more often affected than females; although I have observed many negro women with the disease, I have seen the condition in only one negro man.

Etiology. The etiology of cardiospasm is debatable, although clinical symptoms and postmortem findings suggest that degenerative changes in the vagal nerve ganglia permit overactivity of the sympathetic nerve elements, thus producing continuous spastic contraction of the terminal portion of the esophagus. Lack of a well defined sphincter muscle at the cardia and absence of muscular hypertrophy, except in the wall of the esophagus above the area of stenosis, are factors that have seemed to invalidate cardiospasm as a descriptive term for the disease.

Pathology. The gross pathologic changes observed in the esophagus of patients who have suffered from cardiospasm depend upon duration of symptoms and the degree of dysphagia. In cases of average severity the cardia is tightly contracted and the wall of the esophagus above the area of stenosis is moderately dilated. Portions of the wall may be three or four times thicker than normal, whereas in other areas the wall is much thinner than normal. If there has been little or no retention of secretion and food in the esophagus, the mucous membrane may appear normal; but whenever the esophagus is widely dilated and angulated and secretions have been retained for any length of time, the mucous membrane becomes hypertrophied and ulcerated, and large white macerated areas may resemble malignant degeneration.

Malignant change has occurred in the esophagus in a few cases of cardiospasm, but I have not observed carcinoma at the cardia in any patient who previously had cardiospasm. It is my belief that when carcinoma is found at the cardia in patients with cardiospasm, the original lesion was carcinoma and was confused with cardiospasm. Microscopic studies of the esophagus of patients who have died from cardiospasm, in whom degenerative changes have been demonstrated in the vagal nerve fibers, have not been accepted as authentic

because similar postmortem degenerative changes have been observed in patients who have not had cardiospasm.

Symptomatology.—The three cardinal symptoms of cardiospasm are pain, dysphagia and regurgitation of food. Pain occurs in from 60 to 70 per cent of the cases and varies in intensity from mild epigastric discomfort to attacks of such severity that hypodermic injections of morphine may be required for relief. In location, severity and distribution the attacks may resemble gallstone colic or coronary heart disease. Pain usually begins in the epigastrium and may radiate substernally to the neck, ears and lower jaw, sometimes extending down one or both arms to the elbows.

Often epigastric pain is the initial symptom of cardiospasm and may antedate other manifestations of the disease for months or years. The pain of cardiospasm is usually more frequent and of shorter duration than that of gallstone colic. Absence of jaundice and local soreness over the gallbladder are other features that may be helpful in distinguishing the pain of cardiospasm from that of gallbladder disease.

Even after dysphagia develops, pain usually is not associated with the act of swallowing but occurs at any time, often at night, waking the patient from a sound sleep. Dysphagia is present in practically all patients who suffer from cardiospasm, although sometimes the patient may be unaware that obstruction occurs in the esophagus and may believe that food enters the stomach before being regurgitated. Although varying in intensity, dysphagia is usually continuous from the onset.

The initial difficulty is frequently experienced on drinking cold water, and throughout the disease solid food can be swallowed more readily than liquid. Apples, popcorn and all cold liquids, particularly carbonated beverages, intensify dysphagia. The trouble is usually more pronounced when the patient is eating in a public place or when guests are present in his home. The fear of regurgitation under such circumstances undoubtedly aggravates dysphagia.

At onset of dysphagia there may be loss of weight and strength for a time, but then the patient reaches a level at which the weight is maintained on a restricted diet. A portion of the lost weight may actually be regained if the patient learns to force food through the cardia. This may be accomplished by first taking solid food into the esophagus, drinking a glass or two of water, inspiring deeply with the chin on the chest, and increasing intrathoracic pressure by contracting the thoracic and abdominal muscles without permitting air to be expelled from the respiratory tract. In this manner food can frequently be forced into the stomach in considerable amounts. This procedure is not without risk, however, as in one of my patients spontaneous pneumothorax developed following this method of forced deglutition.

Complete symptomatic obstruction of the esophagus is often noted in cardiospasm, and one of my patients had to take all food through a gastrostomy tube for eighteen years. During this time saliva that accumulated in the esophagus had to be regurgitated at frequent intervals.

At onset of dysphagia food may be regurgitated immediately after it is swallowed or as soon as it becomes obstructed at the cardia. As the disease progresses and the esophagus becomes dilated, food and secretions are retained in the distended sac for a longer time. Sometimes food is regurgitated that has been ingested several days previously. Retention of food in the esophagus may lead to regurgitation and strangulation at night. The patient may not be awakened by the cough associated with strangulation; others in the house may be disturbed, however, and the patient may find that his pillow has been discolored by material he has regurgitated. Regurgitation of blood in small or even large amounts is noted occasionally and unquestionably results from ulceration associated with retention of food in the esophagus.

Pulmonary symptoms are often associated with cardiospasm and may vary from dyspnea after meals, caused by pressure on surrounding structures from a dilated esophagus filled with food, to pulmonary abscess resulting from regurgitation of material from the esophagus and aspiration. Chronic pulmonary suppuration often follows repeated aspiration of food that is regurgitated during sleep.

In a case reported by Shelburne[9] a dilated esophagus filled with food was interpreted as an abscess in the mediastinum on roentgenoscopic examination because in association with regurgitation and aspiration of food, pulmonary infection had developed with fever, cough, leukocytosis and other evidences of suppurative disease. When exploratory puncture of the mediastinal mass revealed material identified as coming from the esophagus, further studies were made and the true nature of the tumor was recognized.

Syncopal attacks have been observed in patients with cardiospasm, and in a few instances sudden death has occurred, evidently from vagal origin. In the patients whom I have observed, the symptoms of cardiospasm have usually been present approximately seven years.

Clinical Findings. In the usual case of cardiospasm general physical examination is essentially negative except for loss of weight that is proportional to the degree and duration of dysphagia. Alteration in the sounds heard over the esophagus with a stethoscope during deglutition were formerly mentioned as significant in the diagnosis of cardiospasm, but probably have little or no value. In severe cases emaciation and dehydration may be pronounced, and pulmonary infection may be apparent.

Studies of the blood may reveal leukocytosis if infection in the lung is acute, or dehydration may result in concentration of all the elements of the blood. Severe hemorrhage from the esophagus is seldom seen in cardiospasm, but when it occurs, secondary anemia may be present.

Roentgenoscopic Examination. If a patient suffering from difficulty in swallowing is in fairly good physical condition and can swallow soft food with a reasonable degree of comfort, roentgenoscopic examination of the esophagus should be made, using a suspension of barium sulfate in water as contrast medium. This may be flavored as desired. If, however, dysphagia is severe and the patient dehydrated and emaciated, roentgenoscopic studies should be deferred until provision has been made for removal of the barium from the esophagus or the patient has swallowed a thread to permit stretching of the area of obstruction. Unless such precautions are observed, barium may complete occlusion of impending stenosis, and gastrostomy for feeding may become necessary.

Roentgenoscopic examination in cardiospasm reveals a smooth cigar-tip type of obstruction at the cardia with more or less dilatation of the esophagus above the point of stenosis. The esophagus may be diffusely dilated and fusiform, S-shaped or flask-shaped. If food is present in the esophagus, areas of irregularity may be noted and may arouse suspicion of malignancy.

Roentgenoscopic study is of value in differentiating other lesions in the esophagus from cardiospasm, especially diverticulum just above the cardia, hiatal diaphragmatic hernia and diffuse spasm of the esophagus.

Special Examination. By chemical and physical methods material that has been regurgitated or aspirated from the esophagus in cardiospasm can usually be distinguished from food or secretion that has been vomited from the stomach.

Esophagoscopy may be useful in the diagnosis and treatment of cardiospasm, but it is not indicated nor desirable in all cases. The necessity for evacuation of all food and secretion from the esophagus during esophagoscopy makes endoscopic study in cardiospasm particularly disagreeable to the patient, and when areas of ulceration are revealed, differentiation from carcinoma may be impossible. Esoph-

agoscopy may be helpful in patients with complete symptomatic obstruction of the esophagus from cardiospasm in facilitating passage of a feeding tube into the stomach, thus obviating the necessity of gastrostomy.

When the esophagus is widely dilated or angulated, the cardia may be difficult to identify. A more helpful method than esophagoscopy in ascertaining that obstruction at the cardia has resulted from spasm is the passage of a No. 60 French sound into the stomach, guided by a previously swallowed silk thread. If there is nothing more than slight elastic resistance at the cardia to passage of the sound, the diagnosis of cardiospasm is practically assured.

Differential Diagnosis. Cardiospasm must be distinguished not only from all lesions in the esophagus that cause dysphagia, but also from lesions that produce acute abdominal and thoracic pain, especially gallstones and coronary heart disease. Secondary manifestations of the disease must also be distinguished from suppurative pulmonary disease and from various lesions that produce attacks of syncope.

Of the lesions in the esophagus that may be particularly difficult to differentiate from cardiospasm, carcinoma is the most frequent and most important. Carcinoma of the cardiac end of the stomach, particularly the scirrhus or "leather bottle" type, may produce smooth obstruction on roentgenoscopic study, and symptoms of dysphagia may have been noted for a year or longer. On passing sounds through the cardia, however, dense obstruction is encountered, which serves to distinguish the lesion from cardiospasm.

Cicatrical stricture seldom occurs at the cardia, and organic stenosis at this area almost always results from carcinoma. Epiphrenic diverticula of the esophagus may cause symptoms that resemble those of cardiospasm, but careful roentgenoscopic study is usually sufficient for identification.

Herniation of a portion of the stomach through the esophageal hiatus in the diaphragm, either so-called congenital shortening of the esophagus with hernia, or para-esophageal hernia, may produce symptoms that suggest cardiospasm; but in hernia gaseous distention is more often observed, and dysphagia is usually of an intermittent type. Roentgenoscopic and esophagoscopic examination is required to determine the type of hernia and to differentiate it from cardiospasm.

"Peptic ulcer" of the esophagus is usually ulceration at the junction of the esophagus and stomach in congenital shortening of the esophagus with hernia and is not a primary lesion. As the area of ulceration heals, contraction frequently occurs and stricture develops. Since the point of stenosis is located just above the diaphragm, hernia with stricture must be differentiated from cardiospasm, which can be done by roentgenoscopic study, passage of sounds through the esophagus and esophagoscopy.

TREATMENT

Medical. Many drugs that act antispasmodically have been utilized in the management of cardiospasm, but for the most part the results of medical treatment have been disappointing. At times pain is sufficiently intense to require hypodermic injection of morphine, but as pain is of a recurring nature, administration of narcotics should be avoided because of the danger of drug addiction.

Belladonna or atropin have been used extensively in an effort to relieve both pain and dysphagia. One of my patients obtained temporary relief from dysphagia by the use of hypodermic injections of atropin sulfate. Increasing amounts of the drug became necessary to produce the desired results until at the time of my observation, $\frac{1}{3}$ gr. was used hypodermically at each injection. After injection of the drug, dysphagia was relieved for an hour or more, during which time food and fluid were ingested in large

amounts. Soon afterward esophageal obstruction became complete again, and all food, fluid and swallowed saliva were regurgitated until a similar dose of atropin sulfate was administered hypodermically. In the majority of patients, relief, even of a temporary nature, is not obtained by antispasmodic drugs. I have given injections of atropin sulfate in doses of $\frac{1}{25}$ gr. every hour until the patient showed evidence of acute poisoning without producing relaxation of the cardia.

Occasionally, the use of tincture of belladonna in doses of 15 to 20 drops every three or four hours has seemed to relieve pain which might persist at the cardia after dilatation of the area of spasm. Nitrites have been recommended in treatment, but I have not seen beneficial results in the few patients for whom I have used this form of medication.

Local treatment of ulceration in the esophagus through an esophagoscope is of no value because ulceration results from stagnation of food, and until relief from obstruction can be obtained, ulceration will persist. Frequent passage of a stomach tube into the esophagus for feeding or lavage is not advisable. One patient, whom I observed, produced severe hemorrhage from trauma in the lower portion of the esophagus by this practice.

When dehydration and emaciation are present, introduction of fluids, usually a solution of normal saline containing 10 per cent glucose, should be given intravenously in adequate quantity. This treatment may be supplemented by subcutaneous injection of fluid and also by proctoclysis. Transfusion of whole blood or blood serum may be advisable in certain patients.

Surgical. Many surgical procedures have been employed in the management of cardiospasm. One of the earliest forms of surgical treatment was the relatively simple procedure of opening the stomach and dilating the cardia manually. In two of my patients this method was used because tortuosity of the esophagus prevented introduction of a dilator from above. When this operation becomes necessary, location of the cardia is facilitated by having the patient swallow a thread prior to exploration, the thread serving as a guide to the cardia for the fingers which are introduced into the stomach.

Plastic operations on the cardia, such as are employed in congenital hypertrophic pyloric stenosis, have also been utilized in cardiospasm but with indifferent results. In marked dilatation and angulation of the esophagus esophagogastrostomy has been performed, but the risk of the operation is high and functional results have not proved satisfactory.

Accumulating evidence that cardiospasm is the result of overaction of the sympathetic nerve supply of the cardia has led to operations designed to interrupt the sympathetic nerve fibers enervating this portion of the esophagus. A few fairly satisfactory results have been obtained by removing sympathetic ganglia in the splanchnic area, whereas other surgeons who believe that the major portion of the sympathetic nerve supply of the cardia originates in the cervical area, have resected the cervical sympathetic ganglia. Probably complete or almost complete removal of the sympathetic nerve fibers that control the contracting function of the cardia would relieve symptoms of cardiospasm, but it is likely that enervation is derived from many areas and that complete sympathectomy is impossible.

Gastrostomy as a palliative operation is seldom necessary and whenever it has been performed, usually indicates ignorance of manifestations of cardiospasm or neglect in treatment.

Dilatation. Mechanical stretching of the cardia has proved the most effective method for treatment of patients with cardiospasm. As indicated under "Treatment"—"*Surgical,*" dilatation of the cardia has been accomplished in a few patients by opening the stomach through an abdominal incision and stretching the area of spasm by introducing several fingers from the stomach into the esophagus. A simpler

and more satisfactory method of dilating the cardia is by introduction of sounds or a dilator that can be distended in the esophagus, through the mouth, guided into the stomach by a previously swallowed silk thread.

The thread should be twisted and not braided silk, and size D is usually small enough to be swallowed easily and yet strong enough to provide sufficient guidance for the dilator. When the thread is swallowed at the rate of about a foot an hour, it will pass through the esophagus into the stomach and coils of small intestine so that after fifteen or twenty feet have been swallowed over a period of twenty-four hours, it can be drawn taut without being pulled out of the stomach. When the thread is thus fixed, it will permit guidance of sounds through the area of obstruction. After the thread has been utilized as a guide and dilatation has been accomplished, the thread outside the mouth is cut off and the portion which has been swallowed will pass through the gastrointestinal tract and be expelled in the feces.

If symptomatic esophageal obstruction is pronounced, the patient may have some difficulty swallowing a thread, and perseverance will be required before a sufficient amount is ingested to permit its use as a guide for passage of dilators. When a patient has failed to swallow a thread after attempting to do so for several days, many ideas may be suggested to facilitate its passage into the stomach, one of which is to tie a shot to the end of the thread. It has been my experience, however, that any method other than the simple procedure of taking the thread on the tongue and ingesting it slowly and continuously is neither desirable nor effective. If a patient has complete obstruction of the cardia and difficulty is encountered in swallowing a thread, esophagoscopy may be necessary in order to introduce a tube into the stomach for feeding. It is advisable, however, to continue to insist for an indefinite period of time that the patient attempt to swallow a thread before esophageal instrumentation is employed.

In case it is found to be impossible to enter the stomach with an esophagoscope, soreness of the throat occasioned by esophagoscopy will further interfere with deglutition and may necessitate gastrostomy for feeding.

After a thread has been swallowed, the cardia should be dilated with sounds. A blunt olive, No. 41 French, is first introduced into the stomach over the guiding thread, and if obstruction is not encountered, it is followed by passage of a No. 60 French sound. Approximately 10 per cent of the number of patients so treated will be relieved temporarily from all symptoms. In half of the patients who obtain relief, symptoms do not recur.

In those who have recurrence of symptoms and in the larger group who do not obtain temporary complete relief from dysphagia by passage of sounds, the cardia should be distended with some type of expanding dilator. For this purpose I prefer the Russell hydrostatic dilator that was made popular by Plummer. The dilator is introduced over the guiding thread into the cardia and is expanded with water, usually to twenty-six or twenty-eight feet of water pressure. This almost always causes pain and a small amount of bleeding, but following dilatation all types of food can be swallowed normally, and in 75 per cent of cases relief from symptoms is permanent. In the remaining cases there may be mild recurrence of symptoms during the six months following dilatation, but in the majority of patients complete relief will be obtained by repeating the treatment. Recurrence almost never happens in a patient who obtains relief for six months following treatment.

Epigastric pain following passage of sounds or stretching with the hydrostatic dilator may be sufficiently severe to require hypodermic injection of morphine sulfate, gr. 1/6 to 1/4. The pain may occur immediately after the cardia has been

dilated or twenty-four to thirty-six hours later. When pain is mild, it may be relieved by sipping hot water, but when it is persistent, 15 drops of tincture of belladonna three or four times a day may afford relief.

Inflation of an expanding dilator with air is not so satisfactory as using water pressure because air is more difficult to control, and adequate pressure with air is not easily obtained. I have not used Hurst's mercury bougies, but I do not believe they can be introduced through the cardia so comfortably, accurately, or safely as sounds guided by a previously swallowed silk thread.

Prognosis. If dilatation of the cardia with a hydrostatic dilator is reserved for patients who have not obtained relief from passage of a No. 60 French sound into the stomach, the danger attending dilatation of the cardia in cardiospasm is negligible. In over a thousand consecutive dilatations of the cardia with sounds and a hydrostatic dilator during the past fifteen years I have not had one serious complication.

Relief from symptoms occurs immediately following treatment, and significant recurrence of discomfort is rarely observed. Patients who require treatment more radical than dilatation are seldom seen. Probably the majority of patients who have been subjected to radical treatment could have been relieved by much simpler procedures. Failure to relieve cardiospasm by hydrostatic dilatation usually results from lack of persistence on the part of the patient in swallowing a thread so that the dilator is not accurately introduced into the cardia or from inability to maintain proper position of the dilator in the cardia during inflation.

DIFFUSE SPASM OF THE ESOPHAGUS AND SPASM AT THE CARDIA

The symptoms of diffuse spasm of the esophagus may be indistinguishable from cardiospasm, but roentgenoscopic study will usually be sufficient for differentiation. In cases of diffuse spasm the lower half of the esophagus presents evidence of excessive spastic contraction on roentgenoscopic examination, and on esophagoscopic examination the lumen of the esophagus may appear almost obliterated. Dilating sounds may be passed through the esophagus, but usually enough resistance is encountered to require a moderate degree of pressure before the sounds can be introduced into the stomach. Relief from dysphagia is less often obtained by dilatation in diffuse spasm than in cardiospasm.

One of my patients, who suffered from diffuse spasm of the lower half of the esophagus, required gastrostomy for feeding for eight years and then was spontaneously relieved from difficulty. During the activity of the spasm he was treated by medicines, esophagoscopy, hydrostatic dilatation of the cardia, passage of sounds, and bilateral cervicothoracic sympathetic ganglionectomy, all without beneficial effect. Two years after treatment was discontinued, symptoms disappeared as suddenly as they had appeared.

In the majority of cases of cardiospasm the disease is not associated with intrathoracic or intra-abdominal lesions and is not dependent upon emotional factors for its origin or perpetuation. Diffuse spasm, however, may be associated with intra-abdominal lesions, and intermittent spasm at the cardia is frequently seen in patients who are nervously unstable.

Intermittent spasm at the cardia is seldom associated with significant symptoms other than epigastric pain and recurring obstruction of food at the cardia. Roentgenoscopic examination usually does not reveal evidence of obstruction at the cardia, but occasionally the passage of a sound will afford relief from symptoms.

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Cyanosis may be defined as a bluish discoloration of the skin and visible mucous membranes. It may vary in tint from a deep purplish hue to a greyish slate color. It is due to the presence of an abnormal amount of reduced hemoglobin in the capillary blood.

DIAGNOSIS OF INTESTINAL OBSTRUCTION*

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THE increase in our knowledge concerning the pathological physiology and mechanical factors involved in patients with intestinal obstruction has changed many of the concepts of proper treatment for this condition. Such changes, however, have placed a premium on a more exact and detailed diagnosis of the individual case. No longer can the surgeon feel satisfied merely to make a diagnosis of "intestinal obstruction" and advise immediate laparotomy. The study of the case should include reasonable efforts to supply information on four additional points: (1) At approximately what point in the gastrointestinal tract the bowel is obstructed; (2) what degree of obstruction is present; (3) whether the obstruction is simple or strangulating in character, and (4) what pathological condition is responsible for the presence of the obstruction.

With proper diagnostic methods these refinements in diagnosis, especially the first three, can be achieved with considerable accuracy. Space will not permit a detailed discussion of the many lesions which may either primarily or secondarily be responsible for the obstruction. While the character of the lesion may be definitely determined in many instances, in others it can only be surmised on the basis of probability.

The importance of an accurate detailed diagnosis can be most appreciated by realizing that the best interests of the patient are not necessarily served by an immediate operation. Only those patients in whom the obstruction is strangulating in character or in whom there is a complete obstruction of the colon demand immediate surgical intervention. Other patients can be satisfactorily controlled for a matter of hours or days, by means of constant suction applied to an indwelling tube, such as the Wangensteen type of Levin duodenal tube or the Miller-Abbott tube. The time thus gained during which the physiological status of the patient can be restored to near normal by the parenteral injection of saline and glucose solutions or the administration of blood transfusions will be appreciated by all surgeons of experience. Such treatment carried out in the preoperative period definitely decreases the risks of operation and has been one of the factors contributing to the lower mortality rates now appearing in the literature. Operation may even be avoided in an appreciable number of patients treated by conservative means. It is of the utmost importance, however, that operation be withheld even temporarily only from those patients in whom there is no complete obstruction of the colon or strangulation of the bowel.

BASIS OF A DIAGNOSIS

The making of any diagnosis is the result of rational mental processes by which an opinion is formulated on the basis of collected evidence. In general, it can be said that such evidence is collected from three sources as follows: (1) The medical history; (2) the physical examination, and (3) the results of special diagnostic procedures: (a) laboratory examinations and (b) *x*-ray examinations.

Medical History. The importance of a complete knowledge of a carefully taken history in making a diagnosis of intestinal obstruction can not be overemphasized. It may serve only to confirm a diagnosis which seems obvious from the gross physical findings, yet many cases occur in which the correct detailed diagnosis can be made

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only after the sequence of events as related by the patient or his attendants are appraised by the physician and integrated with other determined facts. Particular attention should be paid to the following points in the history:

Age of the Patient. Many lesions producing obstruction are suggested by the age of the patient. Meckel's diverticulum and malrotation of the gut are usually seen in children or young adults. Seventy-five per cent or more of cases of intussusception occur before two years of age. Obstruction of the colon which is usually due to a constricting carcinoma is rare before the age of forty.

Nature and Extent of Previous Abdominal Operations. Adhesions within the peritoneal cavity as a consequence of abdominal surgery account for such a large percentage of cases of obstruction that careful inquiry should always be made with respect to the patient's previous illnesses. Vidgoff found that 68 per cent of patients with obstruction in a serious of 266 cases had had previous abdominal operations. Finney and Miller found that 50 per cent and 24 per cent, respectively, of the patients in their reported series had had a previous laparotomy. On the other hand, intestinal obstruction due to adhesions without a history of previous abdominal surgery is rare.

Duration of the Present Illness. Knowledge of this character is important for it permits the physical findings to be considered in their true significance.

Character of Onset of Symptoms. The manner in which the symptoms of intestinal obstruction make their appearance depends largely on the degree of obstruction present. Most cases of acute obstruction exhibit a sudden onset of abdominal symptoms. In cases with partial obstruction the onset of symptoms is more gradual and intermittent. Frequently, a partial obstruction of the colon due to a slowly growing carcinoma suddenly becomes complete. In these cases the intermittent slowly progressive symptoms of partial occlusion become more severe and unremitting. The history usually given in these cases is characteristic.

Character of the Abdominal Pain. The pain of which patients with mechanical obstruction complain is cramp-like in nature with short intervals during which there may be little or no pain at all. These intervals vary from a few seconds to several minutes. If the obstruction is in the colon, the cramps tend to be less frequent than if the obstruction is in the small intestine. If strangulation is present in addition to obstruction, there is constant pain with exacerbations corresponding to the intestinal contractions.

This intermittent pain of obstruction is due to increased peristaltic activity of the bowel. These contractions are extremely severe at the beginning but with the passage of time as the gut becomes more distended it tends to lose some of its power of contraction so that within forty-eight or sixty hours the cramps become less severe and less frequent. At this stage, however, patients with a simple type of obstruction may experience a moderate degree of constant abdominal pain and soreness which may strongly suggest the presence of a strangulation. This discomfort is due to the peritoneal irritation produced by a plasma-like fluid which exudes from the obstructed part of the bowel.

Frequency of Vomiting and Character of the Vomitus. Vomiting, while one of the most characteristic symptoms of obstruction, may be entirely absent if the obstruction be in the colon. This is particularly true in those patients in whom the obstruction in the colon develops slowly. Furthermore, the vomitus in colonic obstructions is scanty in amount and never fecal in character. At most, the patient vomits small amounts of gastric and duodenal contents at infrequent intervals. On the other hand, in patients with obstruction of the small bowel, vomiting is frequent in occurrence and the vomitus copious in amount. The higher the point of obstruction the more severe is the vomiting.

Reverse peristalsis carries the contents of the bowel proximal from the point of obstruction up to the stomach so that soon after the onset of vomiting the vomitus assumes a fecal character as attested by its smell and appearance. With the passage of time as peristalsis becomes weaker and the patient dehydrated, vomiting becomes less severe and assumes a regurgitant character.

Presence of Audible Intestinal Noises. A history of audible intestinal noises associated with abdominal cramps can frequently be obtained in cases with obstruction of the small bowel. Such evidence, however, can only be considered as confirmatory of other diagnostic evidence and must be evaluated by the physician.

Passage of Feces and Gas. The passage of feces and gas after the onset of symptoms suggestive of intestinal obstruction does not necessarily exclude the diagnosis. It must be remembered that the bowel distal to the point of obstruction is normal, both anatomically and physiologically. It will continue its usual functions until it is empty. It can be said, however, that patients with a complete obstruction will not usually expel either feces or gas beyond twenty-four to thirty-six hours after the occurrence of the obstruction. Patients with a partial obstruction usually complain of constipation if the obstruction is in the colon. If the obstructing lesion is in the small bowel, there is usually constipation for a day or so at the time of the recurrent attacks of abdominal pain with normal defecation in the interval.

Recent Loss of Weight and Strength. A recent loss of weight and strength is usually complained of by patients with obstruction caused by a malignant growth. There are, of course, many other causes for loss of strength and weight, but these symptoms are always significant and should be carefully evaluated by the clinician.

PHYSICAL EXAMINATION

A careful physical examination is unquestionably the most important factor in determining whether or not the bowel is obstructed. The history is only suggestive. In addition, the diagnosis of strangulation is dependent largely upon eliciting signs of peritoneal irritation by palpation. Physical examination is of less value in determining the degree of obstruction but the palpation of abnormal abdominal masses frequently furnishes the clue by which the character of the obstructing lesion can be surmised.

General Examination. The attention of the examining physician should be concentrated, for the most part, on the abdomen. Patients with simple bowel obstruction do not, as a rule, look particularly ill in the early stages of their illness. A slight elevation of the pulse rate and temperature may be present. Persistent vomiting, in time, will produce the signs of dehydration and circulatory collapse due to hypochloremia. Such patients have the external manifestations of deep surgical shock. Breathing is rapid and shallow. There is a generalized cyanosis and the skin is clammy with perspiration and cold. The blood chloride level will usually be below 450 mg. per cent.

If the obstruction be a strangulating type, the patient, as a rule, appears to be much more ill from the beginning of his illness. Alarming signs and symptoms of circulatory collapse develop much more rapidly than in patients with simple obstruction and from an entirely different reason. With strangulation the signs of collapse are due initially to the loss of blood into and out of the infarcted segment of gut, but as the bowel loses its viability bacteria and their toxins gain entrance into the peritoneal cavity and superimpose the consequences of a peritonitis. This loss of blood which occurs initially has virtually the same effect as an internal hemorrhage and is produced by the obstruction of the venous blood flow in the involved bowel. As the arterial circulation is rarely obstructed as much as the venous circulation, blood continues to be pumped into the infarcted bowel and escapes from the capillaries until thrombosis occurs. Therefore, it is evident that the severity and

rapidity of development of the initial systemic signs of strangulation tend to vary with the length of bowel involved. The peritonitis which develops in these cases may be localized or masked in certain instances. For example, in intussusception the infarcted intussuscipiens is sheathed by viable bowel and peritonitis is usually seen only in neglected cases. Strangulation occurring in an incarcerated hernia tends to produce a localized peritonitis at first. If these cases are neglected, however, the infection will spread and become general.

The general appearance and signs of a patient with a strangulation obstruction will be, therefore, those of shock. There is no need to enumerate them or the additional signs of peritonitis which appear later.

Examination of the Abdomen: Inspection. Abdominal distention has long been recognized as one of the cardinal signs of obstruction. This is due to the accumulation of gas and fluid in the bowel proximal to the point of obstruction. The degree of distention varies a great deal, being less in cases with lesions high in the jejunum and greatest in lesions of the ileum or colon. Minimal amounts of distention are very difficult to detect in obese individuals. In those cases in which the obstruction is in the transverse or ascending colon, the distention tends to be limited to the right side of the abdomen.

If the patient is not obese, the pressure of distended loops of bowel may give a corrugated appearance to the anterior abdominal wall and the movement of peristaltic waves along these corrugations may be detected from time to time. Such a picture always indicates that the obstruction is chronic in nature. Intestinal loops with normal muscular coats can never be seen. The hypertrophy of the muscle which comes with chronic obstruction must be present.

Palpation. Palpation of the abdomen should be performed with three things in mind: (1) Determine the presence of an abnormal mass; (2) determine the presence of a hernia, and (3) determine whether or not peritoneal irritation is present.

The presence of any tumor should be detected if possible for these lesions are a frequent cause of obstruction. The tender mass produced by an intraperitoneal abscess may explain the etiology of an obstruction due to adhesions. The infarcted bowel of intussusception can not infrequently be felt if the patient can be made to co-operate. When it is realized that hernias by the incarceration of a segment of bowel are the most frequent cause of obstruction, the importance of a careful search for such lesions is self evident.

It is extremely important to determine the presence, extent and degree of any peritoneal irritation. On the basis of this part of the examination alone, the physician is often forced to decide whether or not strangulation is present. Tenderness, rebound tenderness and muscle spasm must all be carefully sought for and considered critically. Unless there is a satisfactory explanation of these findings on other grounds, their existence in a patient with obstruction must always be taken to indicate the presence of a strangulation. In the occasional patient, a moderate degree of peritoneal irritation will be found accompanying a simple type of obstruction. This is due to the transudation of a plasma-like fluid from the walls of the distended gut into the peritoneal cavity. A few patients, therefore, with simple obstruction may be subjected to operation under a mistaken diagnosis of strangulation. This fact, however, should not deter the surgeon from operating without delay in all cases in which the signs of peritoneal irritation are definite.

It has been observed at operation that a simple obstruction, due to an adhesion which is attached to the anterior abdominal wall, is frequently associated with some soreness and tenderness localized to the immediate area of attachment. In these cases, no other signs of peritoneal irritation are usually present. The symptoms are undoubtedly due to the tension of the

adhesion produced by the obstructed bowel.

Auscultation. The careful auscultation of the abdomen with the stethescope is an essential part of the examination never to be omitted. The point to determine is whether or not borborygmi are heard coincident with occurrence of abdominal cramps.

If such a relationship can be established, the existence of intestinal colic is proved. The presence of intestinal colic is strong presumptive evidence that a mechanical obstruction is present. If enterocolitis, food allergy or some dietary indiscretion can be ruled out, the diagnosis of obstruction is not to be questioned. It should be noted, however, that paralytic (inhibitive) obstruction, such as is so universally seen after major abdominal surgery, exists without the close relationship of abdominal cramps and borborygmi noted above. No attempt has been made in this paper to include a discussion of this condition. As previously indicated, after the passage of time the peristaltic activity of the bowel becomes weaker so the borborygmi which may be heard are fainter. The same situation may obtain in cases of strangulation obstruction after peritonitis develops. It should likewise be born in mind that the borborygmi in cases of obstruction of the colon are much less prominent and less frequent than in cases of obstruction of the small bowel.

The auscultation of the abdomen for only a few seconds may be sufficient to determine the presence of intestinal colic, but not infrequently the internal between the abdominal cramps and borborygmi is prolonged. The examiner, therefore, should listen carefully for fifteen to twenty minutes before finally excluding the presence of colic. At times, the gentle massage of the abdomen or having the patient swallow a small amount of water will initiate the cramps and borborygmi.

It can not be said, as others have reported, that there is any characteristic quality of the borborygmi accompanying intestinal obstruction. Many factors play a part in the production of these sounds. Among them are, strength of the peristaltic wave, the size of the distended loop of bowel and the relative amounts of gas and fluid which the bowel contains.

Rectal and Vaginal Examination. Examination by rectum and vagina is important to detect the presence of any masses within the pelvis which may either be the cause of the obstruction or produced by it. In simple obstruction of the small bowel, distended fluid-filled loops of intestine cannot infrequently be palpated by the examining finger. Strangulated blood-filled coils of gut, associated with a diffuse pelvic tenderness, can be perceived more definitely. A carcinoma of the rectum causing obstruction can often be directly palpated. In cases with intussusception rectal examination may reveal the doughnut-like intussuscipiens as an intracolonic mass. In other cases with the same condition, though no mass may be felt, blood can be seen on the examining finger.

SPECIAL DIAGNOSTIC PROCEDURES

Laboratory Procedures. The existence of a simple type of obstruction *per se* produces no characteristic changes detectable by the commonly employed laboratory procedures. If vomiting has been persistent to the point of definite dehydration changes in the blood associated with hemoconcentration, hypochloremia and extrarenal uremia will be found. The hemoglobin concentration, the red blood cell count and the white blood cell count will be elevated. The blood urea nitrogen content and the carbon dioxide combining power of the blood will be increased. The chloride concentration in the blood plasma will be decreased. The urine may or may not show a slight amount of albumin and a few hyaline casts.

The laboratory findings of strangulation obstruction are the result of three factors: dehydration from vomiting, loss of blood from internal bleeding and the development of peritonitis. The elevated white

blood cell count and relative increase in the number of polymorphonuclear leucocytes are the most consistent changes found.

X-ray Examination. The use of the *x*-ray in the diagnosis of intestinal obstruction has become a routine procedure in most surgical clinics. In no instance should a diagnosis be made on the *x*-ray examination alone, but as a means of confirming the results of a physical examination and as a means of excluding other possible diagnoses, roentgenological examination is unexcelled.

The most valuable film is the so-called "scout film" of the abdomen taken with the patient lying prone. The obstructed loops of bowel on such a film are indicated by the quantities of gas which they contain. Because of the distance between the bowel and the film, these gas shadows appear on the film magnified approximately 25 per cent. Some clinicians have advocated taking the film with the patient lying prone and others with the patient sitting upright or standing. In the prone position, the gas shadows form a bizarre pattern due to the displacement of the intestinal loops by pressure of the vertebral column. In the upright position, multiple fluid levels seen scattered throughout the abdomen are found in the presence of obstruction. Neither of these positions gives as comprehensive or as valuable information as a film taken in the supine position.

The information to be sought from the *x*-ray film is concerned with the amount and position of accumulations of gas in the gastrointestinal tract. Normally, gas can be visualized in the small intestine only in infants and small children. The reason for this fact is not clear. Adults and children alike, however, exhibit normally, accumulations of gas in the colon. If there is stasis of the contents of the small intestine, the contained gas and fecal content separate out and the gas is seen as discrete shadows on the film. The visualization of gas contained within the small bowel, therefore, may be taken to mean obstruction in the broadest sense of the term.

The gas accumulations in the small intestine assume characteristic patterns depending upon the portion of bowel involved. The distended ileum appears as an almost smooth tube. The jejunum shows a feathery outline due to the very prominent plicae circulares. Gas shadows in the colon appear coarsely scalloped, due to the haustrations. Familiarity with these patterns and a careful study of the *x*-ray film usually permits an accurate estimation of the site of the obstruction to be made.

The presence of loops of distended small bowel with little or no gas in the colon indicates that the obstruction is probably complete and in the small bowel. The restriction of the visible gas to the colon is the usual pattern found with acute obstructions of the colon. In about 25 per cent of such cases, however, the ileocecal valve is incompetent and allows gas from the colon to pass into the small bowel. In these cases, therefore, the terminal ileum may be distended as well as the colon. Partial obstructions of the small bowel, if at all severe, will exhibit some distention of the proximal bowel with small scattered accumulations in the colon. If the obstruction is in the colon, the visible gas is confined to the colon almost entirely and this is not great in amount. The gas seen on the *x*-ray film of a patient with paralytic (inhibitive) ileus may on occasion simulate closely the gas patterns of any type of obstruction. In the usual case, however, stasis of fluid and gas occurs in both the colon and small bowel. The size of these accumulations depends on the duration and severity of the ileus as well as the quantity of air which the patient swallows.

DIFFERENTIAL DIAGNOSIS

The diagnosis of a typical acute mechanical small bowel obstruction is not difficult. Recurrent crampy abdominal pain associated with increased borborygmi and vomiting if confirmed by an *x*-ray examination can leave little doubt. Occasionally, other abdominal colics must be excluded. This differentiation is best made with the

stethescope and careful auscultation of the abdomen. An acute gastroenteritis sometimes closely simulates an early acute obstruction. However, the vomiting associated with gastroenteritis ceases to be productive when the stomach and duodenal contents have once been removed. Furthermore, diarrhea is usually a prominent symptom of gastroenteritis.

The diagnosis of chronic small bowel obstruction rests largely on the basis of a history of previous attacks and the presence of intestinal colic. The *x*-ray aids in the diagnosis by furnishing for most part "negative" evidence. This type of information, however, is frequently of major importance in arriving at many diagnoses and should not be disregarded.

The existence of peritoneal irritation in a patient with suspected obstruction immediately brings up the question of strangulation. The chief differentiation must be made between strangulation obstruction and inflammatory lesions such as acute appendicitis, acute salpingitis and twisted ovarian cyst. A careful clinical history, pelvic examination and *x*-ray studies of the abdomen are most helpful.

The most difficult decision which the physician has to make in dealing with obstruction is to recognize a strangulation obstruction which develops on the basis of a simple obstruction due to adhesions produced by an acute inflammatory process. Fortunately, this problem does not frequently arise. One can hardly say that all patients with acute obstruction and signs of peritoneal inflammation should be operated upon. Simple obstructions can be adequately controlled usually for days without operation and patients with definite peritonitis are in the main best treated by conservative methods. However, the price to be paid by neglecting to operate upon a strangulation obstruction is so great that in cases in which such a diagnosis cannot be reasonably excluded, the lesser of two evils would seem to be an exploratory laparotomy. A rapid increase in the severity of abdominal pain and tenderness associated with a concomitant elevation of the pulse rate usually attends the onset of strangulation.

If any doubt remains as to the presence of an obstruction of the colon after consideration of the history and physical examination, the *x*-ray film usually settles the issue. A word of caution is in order concerning obstructions located in the cecum and ascending colon. A competent ileocecal sphincter produces a short, closed loop in these cases which may become distended and even rupture without producing alarming symptoms. Since the distended loop is so short, no clinical distention may be detected even on the *x*-ray film, and unless the possibility of such a lesion is borne in mind, the significance of the distended segment may be discounted. Crampy pain with tenderness in the right lower quadrant of the abdomen with or without the presence of a palpable mass in this area should suggest the diagnosis.

Strangulation obstruction involving the colon is almost always due to a volvulus of the sigmoid flexure. Usually these cases exhibit an unusual amount of abdominal distention. Signs of peritoneal irritation will be dependent upon the degree of vascular occlusion occasioned by the twist of the bowel. Many cases give a history of previous subacute attacks. Frequently the pattern of the gas shadows on the *x*-ray film suggests the diagnosis. Fluoroscopic examination and administration of barium by rectum will confirm the diagnosis in a suspected case.

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CARCINOMA OF THE STOMACH*

DIAGNOSTIC ASPECTS

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THE diagnosis of carcinoma of the stomach during the stage in which it is amenable to surgical treatment still presents a serious problem to the clinician, surgeon and radiologist. The principal reason for this difficulty lies in the fact that in the early stages of the disease appreciable symptoms may be lacking, or if they exist, present anything but a definite and clear-cut clinical and radiologic picture. The essence of the problem at the moment, therefore, is to entertain the diagnosis of this disease on suspicion and when such suspicion has been aroused to command every possible clinical, radiological, gastroscopic and even surgical facility to establish its diagnosis. This means that in addition to examination by these methods consideration must be given to careful observation of the clinical course of the patient under suspicion and at times to surgical exploration of the stomach when a lesion of undetermined nature is found to exist within it.

Reasons for emphasizing these points are well brought out in the recent review by Gray, Walters and Priestley on the operability and resectability of carcinoma of the stomach. In their experience at the Mayo Clinic in 40 to 50 per cent of cases the lesion is an inoperable one when it is found on clinical examination and in half of the cases in which operation is performed the malignant process will have progressed to such a degree that nothing of a curative nature can be effected. Furthermore, figures on the resectability of carcinoma of the stomach have shown surprisingly little change over a period of fifteen years according to these observers. It is pointed out that the percentage of patients who underwent resection of the stomach as compared to the total number of patients who had gastric carcinoma has remained consistently near 30 per cent during this period (1926 to 1940).

In any consideration of the diagnosis of carcinoma of the stomach consideration must be given briefly to such problems as the incidence of the disease and the pathologic anatomy of it, for these phases of the problem give to the clinician an orientation which more readily sets him on his diagnostic path.

INCIDENCE

Carcinoma occurs more commonly in the stomach than in any other organ. Eusterman[3] has pointed out that practically 40,000 persons in the United States die every year from this disease. This figure corresponds quite closely with that for the number of persons killed annually in the country by automobile accidents. The disease occurs in men two or three times as frequently as it does in women, and the average age of occurrence is fifty-three years. Approximately 95 per cent of patients with carcinoma of the stomach fall in the age group between forty and seventy, while 5 per cent are less than forty years of age.

PATHOLOGIC ANATOMY

It generally is recognized that there are several pathologic types of carcinoma of

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the stomach and that the disease may occur in any portion of the stomach. The symptoms with which the disease manifests itself depend to a considerable extent on the type of lesion present, the duration of it, and probably even more on the location of the lesion in the stomach. One type is the fairly well localized tumor which proliferates into the lumen and wall of the stomach forming the well known papillary, medullary or cauliflower-type of growth. Such a lesion involving the pyloric end of the stomach may first manifest itself by symptoms of pyloric obstruction and gastric retention and these phenomena may occur while the lesion is still a small one. When this type of lesion occurs in the fundus and especially on its anterior, posterior or greater curvature aspects, it frequently may be silent clinically except for the development of symptoms referable to anemia and cachexia.

Another type of lesion is that represented by diffuse infiltration of a part of or all of the wall of the stomach. This so-called scirrhus or linitis plastica-type of the lesion is the most common form, it is the most difficult to cure and in its clinical manifestation various types of dyspepsia, anorexia and at times obstruction of the pylorus commonly occur. Finally carcinoma, which appears roentgenologically in the form of ulcerous lesions, may pathologically in gross form simulate benign peptic ulcer. Symptoms associated with such lesions not infrequently resemble those of "ulcer-type of dyspepsia" and penetration of the lesion or gross bleeding from it occur also.

Another point of considerable interest in the pathologic anatomy of this lesion is in relation to its pathogenesis, particularly as it relates to the previously existing state of the gastric mucosa. Many clinicians are of the opinion that carcinoma rarely if ever develops in a gastric mucosa which is normal. The rôle of gastritis and particularly atrophic gastritis as a predisposing factor in the development of gastric carcinoma is widely but not universally accepted.

CLINICAL SIGNS AND SYMPTOMS

Many physicians think of carcinoma of the stomach in terms of the far advanced picture of the disease. From the standpoint of effective treatment it is too late to make the diagnosis when the patient presents persistent indigestion, coffee-ground vomitus, marked anemia and loss of weight, a large palpable abdominal mass and signs of pyloric obstruction. The clinician must be taught to realize that he should attempt to make the diagnosis of carcinoma when only the vaguest symptoms are present if he is substantially to reduce the mortality rate. Any person, and especially one over the age of thirty five, in whom symptoms of indigestion persist over a few weeks without known cause is entitled to a thorough investigation of his gastrointestinal tract. Too often under these circumstances the physician treats the patient symptomatically for a period of months before it becomes apparent that a carefully planned gastrointestinal study should be carried out. Moreover, not infrequently such symptomatic therapy affords the patient temporary relief while an unsuspected cancer silently proliferates. Later when the lesion is found it often is too late to carry out any operative procedure which will afford a permanent cure.

As Bloomfield has pointed out, it is really useless to try to give any composite description of the symptoms of gastric cancer since they are so variable and not infrequently by the time the very first symptom may manifest itself, roentgenologic study may reveal the presence of a large, proliferating tumor which is beyond the hope of resection. However, there are certain characteristic ways in which the symptoms may begin. As previously pointed out (Wilbur) the modes of onset of symptoms of carcinoma of the stomach depend in a large part on the pathologic type and situation of the lesion and to some extent on the reactivity of the individual; that is, whether he is hypersensitive or hyposensitive. Lesions which exist near the pylorus will more likely lead to ob-

struction, whereas those high in the fundus of the stomach will more often produce anemia, loss of weight and strength and vague dyspepsia.

Associated with the most common mode of onset of the disease is the so-called typical history. The characteristics of this typical history are as follows: the patient, usually middle-aged, gives a life long story of perfect digestion until the gradual or rapid onset of persistent dyspepsia, which is characterized by anorexia, fullness and discomfort after meals, belching, burning, nausea and occasionally by vomiting with loss of weight and strength. The story usually is a progressive one, and not infrequently the patient is somewhat emaciated and on rare occasions accompanies his story with the statement that he may palpate an abdominal mass. Not infrequently these symptoms begin following a brief period of illness characterized by the patient as an attack of the "flu."

Carcinoma of the stomach may begin with symptoms like those of peptic ulcer. The symptoms may be of long or short duration, but it should be pointed out that this does not mean that the carcinoma developed in a benign ulcer. The occurrence of symptoms suggesting ulcer may mislead one from the correct diagnosis. According to Eusterman and Kirklin about a fourth of resectable carcinomas of the stomach simulate peptic ulcer, especially at the onset. When a patient past middle age presents a history of ulcer type of dyspepsia of short duration one should be on the look-out for carcinoma. Similarly, suspicion should be aroused if there is loss of weight and strength out of proportion to the reduction of intake of food. When the history with ulcer characteristics is of long duration one should suspect the development of a complication, of associated disease, or of carcinoma, particularly if there occur (1) disappearance of intermittency of exacerbation of symptoms with substitution of a continuous or remittent clinical course, (2) irregularity, diminution, or disappearance of pain-food-ease sequence, (3) substitution of the usual distress by a dull ache, more or less continuous, and aggravation rather than ease by alimentation, and (4) nausea and anorexia. In addition, if carcinoma of the stomach is present the following objective phenomena may be found: (1) lowered gastric acidity, (2) anemia without gross bleeding, (3) occult blood in the gastric contents, and (4) disturbances in motor function of the stomach (Eusterman).[3]

Carcinoma of the scirrhous or polypoid type occurring near the pylorus may produce pyloric obstruction early or late in the course of the disease. When symptoms of obstruction arise early, and especially when they are associated with a previous history of ulcer, diagnosis may be difficult if not impossible. One may have to be content with the diagnosis of obstruction at the outlet of the stomach. As Eusterman[3] has pointed out, gastric retention may occur in cases of carcinoma of the stomach without a lesion at the pylorus, and one should think of this diagnosis when there is a residuum in the fasting stomach if barium is not retained after six hours. In addition, he has pointed out that in 60 per cent of cases in which the lesion is resectable and in 40 per cent of cases in which the lesion is small gastric retention is present.

Anemia is one of the commonest findings in malignant disease. This is particularly true of malignant disease of the gastrointestinal tract, and anemia at times is the first and presenting symptom of carcinoma of the stomach. The anemia usually depends on the presence of an ulcerating or polypoid lesion. If this lesion is in such a position in the stomach as not to interfere with its emptying, anemia may be present before any other symptom develops. The anemia usually depends on bleeding, which is the result of ulceration into vessels or of necrosis and sloughing of small vessels in the growth. It may be sustained in part by poor nutrition of the individual because of the carcinoma, or it may possibly result from a disturbance in the metabolism of hematopoietic substances (intrinsic factor

or pernicious anemia). Carcinoma of the stomach rarely produces gross bleeding except as a terminal phenomenon. In only 1 per cent of cases does it lead to gross hematemesis (Rivers and Wilbur).

Patients who present themselves because of a general decline in health or because of a general weight loss and strength not infrequently have carcinoma of the stomach even though gastrointestinal symptoms are absent. Not uncommonly symptoms may begin with an illness interpreted by the patient as an acute infection of "the flu." Anorexia may be the only symptom, or pallor and weakness alone may be present. It is important to recall that carcinoma of the stomach may develop in the presence of any chronic disease. In the presence of these clinical symptoms the carcinoma is not infrequently situated in the fundus or cardiac portion of the stomach, where it may be difficult to find roentgenologically. Occasionally, specific nutritional disturbances will occur as a result of carcinoma of the stomach. Eusterman and O'Leary have emphasized the development of pellagra in association with gastrointestinal disease, particularly those producing obstruction.

Rarely will the outstanding symptoms presented by a patient with carcinoma of the stomach be the result of metastasis. Metastasis generally does not occur early, but occasionally it may be extensive when the original lesion is small. The usual sites of metastasis, the liver, regional lymph-nodes, peritoneum, pancreas, pleura and lungs are not commonly involved early enough to provoke symptoms before those produced by the initial lesion. If metastasis produces the presenting symptoms, the lesion in the stomach is most likely in a "silent spot" and the metastasis involves a very important organ such as the brain, spinal column or bone marrow.

Physical examination of the patient suspected of having early carcinoma of the stomach generally is not very helpful in establishing the diagnosis. However, unexplained pallor or pasty color, obvious emaciation, or any change in the general appearance of the patient from that of previous good health are suggestive signs. Occasionally, there may be slight tenderness in the epigastrium on palpation; usually a mass is not palpable. When the disease becomes more advanced evidences of pallor, weight loss, ascites and abdominal masses may appear. In all instances the physician should remain constantly on the lookout for evidence of metastasis in the left supraclavicular region, on the rectal shelf and occasionally at the umbilicus.

Ordinary *laboratory procedures* generally are not very helpful in pointing the way to the diagnosis in early stages of the disease. Examination of the blood may, of course, provide evidence of anemia, but frequently the results are normal. Analysis of the gastric content may be helpful in establishing the presence of gastric retention which should lead to further careful investigation, and it may, after stimulation with histamine, reveal a secretion which is less than the normal in volume and acid content. The large number of reports on the character of the gastric secretion in carcinoma of the stomach may be summarized by saying that in general in this disease there is a tendency for the volume of gastric secretion to be low and for the acid content of it to be low or absent. However, in over 50 per cent of cases of early lesions free hydrochloric acid may be present in the gastric content and in some patients with extensive carcinoma of the stomach the volume and concentration of acid may be exceedingly high and resemble those of benign ulcer. While statistical studies of this problem show definite trends in one direction or another, the important clinical point is that such studies fall down completely when an attempt is made to put them to diagnostic use in an individual case. It seems reasonable to conclude that when achlorhydria is found further studies by roentgenologic and gastroscopic methods are imperative.

Examination of the stool for occult blood and observation of blood in the gastric content may be of considerable interest, but generally the results of such examina-

tions are not of great diagnostic importance unless they repeatedly are positive under conditions of careful control of a meat free diet and in the proved absence of other lesions.

ROENTGENOLOGIC DIAGNOSIS

The most reliable widely available diagnostic procedure in the clinical diagnosis of cancer of the stomach is roentgenologic examination of the stomach. In the hands of the skilled roentgenologist this method of examination has rapidly become a highly satisfactory and accurate one, and is in fact worth more than that of all other laboratory methods combined because it is a direct method of examination, it permits visualization of the gastric outline and of the peristaltic and motor activity of the stomach which are invaluable in determining the condition of the organ. The radiologic features of gastric carcinoma are well known and it is not the purpose of this clinical discussion to undertake a prolonged discussion of the characteristic diagnostic aspects of these lesions. In brief, the presence of persistent filling defects, of areas of thickening of the gastric wall with associated rigidity and absence of peristaltic activity, of large and often fixed ulcerating lesions, and of smaller ulcerating lesions especially if they are located on the greater curvature and occur near the pylorus—all are indicative of gastric neoplasm. Roentgenologic examination is of value also not only in detecting the presence of a lesion but it also gives the physician a knowledge of the location of the lesion in the stomach. One of the most commonly debated problems among radiologists and clinicians has to do with the benign or malignant nature of an ulcerating lesion. Experienced radiologists become very skilled in differentiating between such lesions by observations of the size and location of them, the shape and depth of them, the presence or absence of tenderness and associated spasm, and the observation of Carman's meniscus sign. However, the radiologist is not always able to characterize the nature of an ulcerating lesion, and in such cases it is necessary to depend on the subsequent clinical course of the lesion, on gastroscopic observations or on surgical exploration.

Perhaps of greatest importance in considering this subject is that of knowing when to demand roentgenologic examination. No arbitrary rule can be established in regard to the advisability of making *x*-ray studies but it is a good clinical rule to do so when the patient presents symptoms of persisting dyspepsia or those previously noted in the paragraphs on modes of onset of symptoms of the disease. In other words, the examination must be made more on clinical suspicion of the disease in the future if earlier lesions are to be observed. Moreover, if the study is negative and the patient's symptoms persist, further examination should be carried out in four to six weeks.

GASTROSCOPIC EXAMINATION

In recent years observations of the stomach through the gastroscope have provided an additional useful means of diagnosing carcinoma of the stomach. The usefulness of this procedure depends almost entirely on the experience of the gastroscopist, not only in his previous observations of lesions, but also in the correlation of his gastroscopic observations with those of the same lesions at the operating table or by the pathologist. Gastroscopic examination may confirm the roentgenologic diagnosis of the carcinoma and not infrequently it may provide definite evidence of the benign or malignant nature of a lesion subject to questionable roentgenologic interpretation. Occasionally, the gastroscopist is able to see a lesion which is suspected clinically but which for various reasons may be overlooked by the radiologist. Polypoid tumors generally are easily distinguished while ulcerating lesions present a less certain diagnostic problem. The carcinomatous character of a lesion may be suspected when the edges of it are heaped up and fade gradually into the normal gastric mucosa, when the edges of the

lesion are somewhat irregular and the floor of it is covered with necrotic tissue and exudate dirty gray, brown or red in color.

Schindler points out that the importance of gastroscopy in the early diagnosis of gastric carcinoma has been disputed although many gastroscopists recognize its value in the differential diagnosis of benign and malignant lesions and in the determination of operability. He further adds that the early diagnosis may be possible if each patient over thirty-five years of age who suffers from mild digestive symptoms or loss of weight otherwise unexplained is examined roentgenologically and gastroscopically without delay. Gastroscopy may be helpful in the determination of the operability of gastric carcinoma for the extent of the lesion may be more apparent than it is on roentgenologic study.

Schindler follows Borrmann's classification of gastric carcinoma, which is composed of four main types: Type I, polypoid carcinoma, is a sharply limited, operable lesion and carries an excellent prognosis. It was observed by him gastroscopically in 2.9 per cent of carcinomas. Type II, noninfiltrating carcinomatous ulcer, is a lesion which is surrounded by a sharply limited wall and carries a good prognosis. It was found in 17.6 per cent of carcinomas observed by Schindler. Type III, infiltrative carcinomatous ulcer, is a tumor only partially surrounded by a limiting wall, and even if operable the prognosis is still doubtful. This type of lesion occurred in 16.3 per cent of carcinomas. Type IV, diffuse infiltrating carcinoma, need not be extensively ulcerated. It almost always gives a bad prognosis, and even if resectable goes on to early recurrence and metastases. It occurred in 63.2 per cent of the carcinomas observed by Schindler. The gastroscopic picture has been found more characteristic than that of the gross specimen at times, on account of the presence of circulating blood. However, Schindler emphasizes the point that gastroscopy and roentgen-ray examination are not competitive, but that each supplements the other.

CLINICAL COURSE

In an occasional patient, and particularly in that group of patients presenting an ulcer in the stomach, it may be advisable and necessary to follow by close observation the clinical course of the patient and his lesion. The old discussion of whether or not a benign gastric ulcer will become malignant and, therefore, should be surgically removed is of no value whatever in considering the problem presented by an individual patient with such an ulcerating lesion. The important point to consider at the time the patient is observed is whether or not the lesion is malignant. There are only two ways in which the differentiation can be made. The first is to wait and see what happens and if the patient later develops symptoms of extensive cancer of the stomach one can be reasonably sure that at the time of initial examination the lesion was a malignant one. This method obviously is impractical. The second method is to excise the lesion and study it under the microscope for evidence of carcinoma. The gastroscopist would surely add that his method of examination should be included as a third method by which such differentiation can be made. In the hands of the expert gastroscopist this occasionally can be done but there is no gastroscopist whose opinion in this regard is infallible.

From the practical point of view it is not reasonable to operate immediately upon every patient with an ulcer in the stomach; nor is it advisable to ignore the clinical course of such lesions until a later time when it is obvious that the lesion is a benign or a malignant one. The obvious answer is carefully to watch the course of the lesion under medical treatment. This should be done by clinical, radiologic, and when possible gastroscopic means. In a recent review of the subject Eusterman[2] points out that insufficient stress has been placed in the differential diagnosis of carcinomatous and benign ulcerations on the diagnostic significance of the response to medical treatment.

Gradually, however, physicians are accepting the viewpoint that the degree of demonstrable anatomic change in an ulcer undergoing treatment is of as much or greater significance as symptomatic improvement. The importance of this viewpoint is in the fact that the symptoms arising from gastric cancer of all types may be relieved considerably in a large number of patients, but that generally anatomic improvement in the lesion indicates its benign character while failure to heal is indicative of a malignant process. This viewpoint cannot be completely accepted.

The failure of a lesion to heal under medical treatment is not indicative that it is malignant and the apparent healing of the lesion is not proof of its benign character. It is quite true that the majority of benign gastric ulcers will decrease in size or heal on medical treatment and that the large majority of carcinomatous ulcers or ulcerating carcinomas will decrease little in size, if at all, under the influence of medical treatment. Exceptions exist. Another confusing factor is that in recent years the gastroscopist has produced evidence that an ulcer still may be active and unhealed even though there is complete disappearance of the niche from a roentgenologic point of view. And to add further confusion to the general picture, Eusterman[2] recently has reported five cases in which gastric carcinoma not only masqueraded as benign ulcer but reacted to treatment in a similar fashion as well.

Under circumstances of this almost completely bewildering set of observations, what is the average practising physician to do in the handling of his patient with a suspicious looking ulcerating gastric lesion? Eusterman[2] is of the opinion that suspicion of cancer is justified under the following circumstances: "location of the lesion in the prepyloric first inch, posterior wall or greater curvature, large size, consistent achlorhydria after stimulation with histamine, persistent or frequent recurrence of occult blood in the feces during treatment, late onset in an elderly person and reduced gastric acidity and secretory volume estimated under basal conditions, especially in association with early pyloric obstruction." If these symptoms or findings exist then great care must be given if the patient is placed on medical treatment and the criteria so well formulated by Jordan should be followed. These criteria are that while under careful medical treatment there shall be complete disappearance (1) of occult blood from the stool; (2) of radiologic signs of the defect and (3) of all symptoms. To this she now adds a further criterion that the lesion must not recur under treatment. In this connection the importance of gastroscopic observation of a lesion under treatment is very desirable. Taylor on the basis of repeated gastroscopic observations has well stated the value of this procedure when he expressed the opinion that failure of an ulcer to change at all after three weeks of intensive treatment in the hospital is sufficient evidence that it is malignant. Perhaps the statement should be modified and stated as a clinical rule that in the case of such a lesion surgical exploration is justified and in fact demanded on the strong presumption that the lesion is a malignant one.

For those clinicians who do not have an experienced gastroscopist available and perhaps for those who do, the advice of Eusterman[2] is well taken in considering the clinical course of an ulcerating gastric lesion. He states that all such patients "submitted to treatment should undergo routine examination at intervals of three months for at least a year and at intervals of six months thereafter for an additional period of twelve to eighteen months." Such examination should certainly include roentgenologic observations.

SURGICAL EXPLORATION

In the early diagnosis of carcinoma of the stomach, surgical exploration must be considered an important method of diagnosis in some cases. As already pointed out in recent years, despite all the refinements of old and the developments of new

methods of examination of the stomach, in the experience of surgeons[6] at the Mayo Clinic the percentage of patients who underwent resection of the stomach to the total number of patients who had gastric carcinoma has remained consistently near 30 per cent for fifteen years (1926 to 1940). It seems reasonable to conclude among other things that surgeons of a previous day were as successful as we are now in "spotting" such lesions not because they often made the clinical diagnosis preoperatively but because they operated often on the basis of suspicion of the character of the lesion and because they operated more often than is being done now on patients with "gastric ulcers."

It still is good advice seriously to consider exploration for patients who have gastric lesions of unexplained nature, particularly when there is available a surgeon with experience in dealing with this type of lesion.

SUMMARY

If the results of surgical therapy of carcinoma of the stomach are to be effectively improved in the next few years, the diagnosis of the lesion will have to be made earlier. The essence of this problem at the moment is to entertain the diagnosis of the disease on suspicion and when suspicion exists to command every possible clinical, radiological, gastroscopic and even surgical facility to establish the diagnosis. If no more than suspicion of the nature of the lesion exists but the suspicion for good reason persists, the stomach should be explored surgically.

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COMPLICATIONS ASSOCIATED WITH APPENDICITIS*

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IN a discussion of postoperative complications Cutler and Scott[1] state that, "With the period that follows operative intervention begin the trials and tribulations of the surgeon." This statement is probably nowhere more true than in the case of those patients who require surgical attention because of a septic appendix, and in whom infection has extended beyond the confines of this organ. It is a well known fact that today the mortality of appendectomy for uncomplicated acute appendicitis is almost negligible, whereas in the more advanced cases, when peritonitis of some degree has supervened, the mortality continues to be disturbingly high. For the most part, deaths from acute appendicitis are due to some phase of peritonitis either directly, as from toxemia, severe sepsis and ileus, or indirectly from the later complications of peritonitis, e.g., intestinal obstruction, empyema, pneumonia, etc. If one consults the records of the surgical clinic in any large hospital, the more serious complications of acute appendicitis will be found to include general peritonitis, residual or secondary abscesses, intestinal obstruction, major wound sepsis, pneumonia, pylephlebitis, liver abscess, empyema, pulmonary embolism, fecal fistula, postoperative hemorrhage and thrombophlebitis. The present discussion is concerned with the abdominal complications encountered in the management of the perforated appendix and, in particular, with those which occur following operative intervention.

GENERAL PERITONITIS

Diffuse peritonitis is most often the result of sudden perforation in the obstructive type of acute appendicitis which, according to Allen,[2] is responsible for 95 per cent of the deaths from this disease. The obstruction of the appendix is usually due to a fecalith. The process advances rapidly due to interference with the blood supply of the appendix and consequent necrosis of its wall. Perforation may occur as early as four hours following the onset of symptoms. Due to the sudden flooding of the unprepared peritoneum with septic material, little opportunity is afforded for the usual walling-off process to take place and a diffuse or general peritonitis ensues. Coller and Potter[3] found eighty-eight cases of general peritonitis among 336 patients with acute appendicitis, in all stages of the disease, admitted to the University of Michigan Hospital over a three-year period, an incidence of 22.2 per cent. Stafford and Sprong[4] found 196 instances of general peritonitis in a similar group of 1,317 patients admitted to the Johns Hopkins Hospital over an eight-year period. The incidence of peritonitis in this group was, therefore, 14.9 per cent. Barrow and Ochsner[5] found 179 cases of general peritonitis in a group of 1,039 cases of acute appendicitis admitted to the Charity Hospital in New Orleans, an incidence of 17 per cent. The diagnosis of acute appendicitis with general peritonitis is not difficult. The patient complains of severe generalized abdominal pain, with nausea and vomiting. The abdomen becomes distended, tense and rigid. There is tenderness throughout, although it remains more marked in the right lower quadrant. Auscultation reveals a silent abdomen. The temperature is considerably higher (101°F.) than in uncomplicated acute appendicitis, and the pulse rate is increased accordingly. The leucocyte count is also

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high, e.g., 25,000 to 30,000. If the infection continues unchecked, the unmistakable picture of a terminal peritonitis develops. The pulse becomes rapid and thready, there is profuse diaphoresis, the skin is cold and clammy, abdominal distention is marked, the eyes are sunken, the expression is anxious and there is pallor and cyanosis, signs which denote impending death. It goes without saying that the most important treatment is prophylactic, i.e., recognition of the disease and removal of the appendix before infection has spread beyond this viscus. The management of the patient with general peritonitis remains a controversial matter. Many authors, (Horsley,[6] Herrick,[7] Davis, Stafford and Sprong[8] and others), advise prompt operation regardless of the stage of the disease. It is our opinion that these patients with widespread or general peritonitis are best treated by delayed operation and we recommend the Ochsner regimen in order to improve the patient's general condition and to allow localization of the infection to take place before any operative procedure is performed. However, we agree with Allen and his associates that if one sees these patients within four to six hours from the time of perforation, prompt operation is best. By means of a careful history, very often the time of perforation can be determined. There is often an acute increase in the severity of the pain followed by a period of temporary relief. In such instances, while there is soiling of the regional peritoneum and perhaps an early local peritonitis, the peritoneum is capable of handling this limited amount of infection, provided further contamination is prevented by removal of the appendix. Moreover, protective adhesions have not yet formed so that harm does not come from an operation in this stage.

As a postoperative complication, general peritonitis must be regarded as one of the most serious since the mortality is always high. From their study of the literature, Ochsner, Gage and Garside[9] concluded that peritonitis may be the cause of death in as many as 77.5 per cent of patients dying of appendicitis. Fortunately, general peritonitis is not one of the common postoperative complications. When it does occur, it may be due (1) to contamination of the peritoneum by transperitoneal drainage of abscesses, (2) to failure on the part of the surgeon to drain when drainage is indicated at the time of the original operation, or (3) to an ill timed operation whereby early adhesion formation is interfered with; and what would be a localized infection if let alone, is converted into a spreading peritonitis. Occasionally, because of an especially virulent type of infection, general peritonitis will supervene regardless of the time and type of the original operative procedure. The subject of drainage is again a controversial matter. The present day trend is to drain less frequently than formerly, especially in cases in which a diffuse peritonitis is found at operation. However, the axiom, "When in doubt, don't drain," is capable of doing harm in the hands of young or inexperienced surgeons. Hanford[10] gives the following indications for drainage:

(1) Any appendiceal abscess. With this statement practically all authors agree. Drainage is also advised when tissue of doubtful viability must be left in the abdominal cavity, e.g., (a) necrotic tissue, such as part of the wall of an abscess; (b) part of the appendix; (c) the whole of the appendix; (d) damaged tissue, such as the meso-appendix stump, which might become necrotic in the presence of infection.

(2) In acute diffuse peritonitis with creamy purulent or fibrinopurulent exudate of considerable amount or with a malodorous exudate. Some authors would disagree with this statement.

(3) Insecure or doubtful closure of the cecum or the appendix stump.

(4) An unrecovered loose fecalith.

(5) Gross soiling by cecal contents.

(6) Infection or contamination of the retroperitoneal tissue as in a retrocecal appendix.

(7) Uncontrolled bleeding or doubt as to the control of bleeding.

As previously mentioned, general peritonitis may be the result of operative intervention which interferes with the walling-off of a local peritonitis following perforation. In the case of a slow perforation, as in the inflammatory type of appendicitis, the infection is efficiently localized if given a chance. In these slow perforations, the defensive mechanisms of the peritoneum come into play and prevent spread of the process by limiting the infection to the right lower quadrant or pelvis. Loops of intestine and the omentum become adherent to the cecum and appendix and adhesions quickly form. An abdominal mass may become palpable, although this need not be an abscess in the strict sense of this term. The process terminates by progressing to the stage of suppuration or, more often, by spontaneous resolution. Thus, in cases seen during the third, fourth or fifth day of the disease, with a mass present or in the stage of development, operation is best deferred. If performed during this so-called "danger period," Love[11] points out that protective adhesions are broken down and a spreading or general peritonitis may thus be provoked. Moreover, removal of the appendix under these circumstances presents technical difficulties and is attended with the danger of injury to the cecum or intestine. Excessive bleeding may be encountered and often the procedure is prolonged and an undue amount of trauma is inflicted. Simple drainage, on the other hand, is of little value and corresponds to attempts at incision and drainage of a superficial cellulitis while still in the stage of brawny induration. By delaying intervention until the proper time, extraperitoneal drainage of an abscess is possible or, in the case of spontaneous regression of the mass, a clean appendectomy may be performed at a later date.

Spreading peritonitis induced by or following operation may present a variable clinical picture. In young and otherwise healthy adult patients, the usual picture of general peritonitis described above may be observed. However, in elderly persons or those weakened by their infection, fluid and salt imbalance or by a prolonged operation involving excessive trauma and hemorrhage, spreading or general peritonitis may exhibit a very atypical course. Here the usual signs of peritonitis are apt to be far less striking than usual. There is commonly some abdominal discomfort and distention, associated with absence of peristaltic sounds. Nausea and vomiting are common. The temperature may be high or, again, may be but slightly elevated. The pulse is rapid and later becomes thready and uncountable. Therefore, in any patient who fails to do well following operation, the possibility of a general peritonitis should be considered and when suspected the usual regimen (Ochsner) for the treatment of peritonitis should be instituted. This consists in Fowler's position, hot applications to the abdomen, nothing by mouth, liberal quantities of morphine, duodenal syphonage, intravenous fluids, glucose and sodium chloride, blood transfusions, oxygen and drugs of the sulfonamide group. Secondary operations are rarely indicated, as the outcome depends chiefly upon the resistance of the patient and the virulence of the infection. As in any other case of peritonitis treated by the Ochsner regimen, careful vigilance is necessary in order to detect local abscesses as they develop. When present, these should, of course, be drained at an early date.

SECONDARY OR RESIDUAL ABSCESSES

Secondary intra-abdominal abscesses are prone to occur in certain fossae or regions of the peritoneal cavity. These abscesses may represent a favorable termination in cases of general peritonitis which have been treated conservatively and in which the patient has succeeded in overcoming his infection. In our experience, about 60 per cent of the patients with general

peritonitis treated by the deferred operative method, develop abscesses. Similarly, abscesses may be encountered in cases of general peritonitis which have been treated by prompt operation and they may develop regardless of whether or not drainage was instituted and whether or not the appendix was removed. Moreover, they may occur as a postoperative complication following drainage in cases of a localized appendiceal abscess and here, again, regardless of whether or not the appendix was removed. It is for this reason that contamination of the uninvolved peritoneum should be avoided, and that extraperitoneal drainage should be performed whenever possible. Rarely such abscesses may develop as a complication of appendectomy when only a local peritonitis was present at the time of operation. C. W. Cutler, Jr.,[12] in a study of 1,651 patients with acute appendicitis operated upon at the Roosevelt Hospital, found secondary abscesses which required further surgery in thirty-three patients. Such abscesses were found in 4.5 per cent of all cases of suppurative appendicitis. In this group of thirty-three cases, about 60 per cent followed general peritonitis, whereas the remainder occurred as a complication of suppurative appendicitis with local or spreading peritonitis. In his series the mortality was 21 per cent in the entire group, or 13 per cent if the subphrenic abscesses were excluded.

As shown in Figure 1, the common sites for these secondary or residual abscesses are (1) the pelvis, in which case the abscess occupies the cul-de-sac of Douglas, (2) the left lower quadrant, (3) the subdiaphragmatic spaces, and (4) the ileocecal region. True residual abscesses following general peritonitis are often found either in the pelvis or in the subphrenic spaces. This is due to the fact that with the patient in the recumbent position, the marked anterior convexity of the lumbar spine tends to deflect purulent exudates upward or downward, allowing collections to form in the more dependent or isolated recesses of the peritoneal cavity. For the same reason, residual abscesses in the midabdomen are uncommon.

The presence of a secondary intra-abdominal abscess is often suggested by the fact that following operation fever persists, or subsequent to a fall to normal, it suddenly becomes elevated several days later. Along with this, the patient experiences a certain amount of abdominal pain or discomfort, the location of which may be of some diagnostic value. If the process continues for a considerable time, the temperature curve exhibits septic swings and the patient appears worn and haggard from the effects of the continued sepsis. He feels weak and exhausted, has anorexia and malaise and later may develop a secondary anemia. Physical examination reveals localized tenderness on abdominal, rectal, or vaginal examination and a mass may be palpable. Infections in the subphrenic spaces are more difficult to detect and require special attention and study.

Pelvic Abscesses. Abscesses situated in the rectovesical or recto-uterine pouches are the most common of the residual abscesses. In fact, this is the most desirable location for such abscesses and one of the advantages of the Ochsner regimen in the treatment of peritonitis, either independently or in conjunction with operation, is the assistance which it renders in localization of the general infection in the pelvis. With the patient in the recumbent position, the cul-de-sac becomes the most dependent portion of the peritoneal cavity. Whether the factor of gravity alone is important in allowing exudates to settle in the pelvis is problematical. Nevertheless, Fowler's position is one of maximum comfort for the patient and when abdominal distention is marked, it relieves pressure on the diaphragm and thus diminishes respiratory embarrassment. The old view that absorption is less rapid from the pelvis than from the diaphragmatic peritoneum, is no longer tenable. In the pelvis, however, there is a minimal amount of movement, whereas in the subphrenic spaces, localization is interfered with through the constant mo-

tion of the diaphragm. All agree that localization of infection in the pelvis is much to be preferred to localization in the subphrenic or subhepatic spaces, since in the pelvis recognition of an inflammatory mass or abscess is far easier and, also, since here surgical drainage, should it become necessary, is a simpler procedure. It is common experience that the mortality of pelvic abscesses is lower than that of abscesses situated in the upper quadrants of the abdomen. As in the case of localization of infection at any other point in the peritoneal cavity, the presence of an inflammatory mass or area of infiltration does not necessarily denote a true abscess. It is only when suppuration has taken place that surgical drainage is indicated. The majority of these inflammatory masses will undergo spontaneous resolution under proper conservative treatment and such treatment should first be given a thorough trial. The principles involved are essentially those entailed in the treatment of general peritonitis. Restriction of all food and fluid by mouth and the liberal use of morphine are among the most important items in this regard. Gastroduodenal suction and the intravenous administration of solutions of glucose and sodium chloride are valuable adjuncts. Under conservative therapy, over one-half of such pelvic masses will subside and no operative intervention will be necessary.

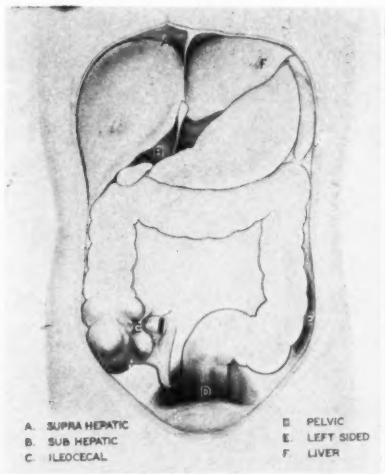


Fig. 1. Diagrammatic sketch showing the more common locations of the residual or secondary intraperitoneal abscesses associated with appendicitis. (Redrawn from Ochsner, A., Gage, I. M. and Garside, E. The intra-abdominal postoperative complications of appendicitis. *Ann. Surg.*, 91: 544, 1930.)

Fig. 2. Diagram showing method of confirming diagnosis of abscess in the rectovesical pouch. If pus is obtained on aspiration, drainage by rectum is then instituted.

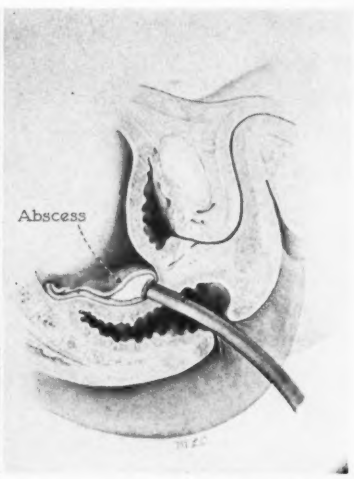


Fig. 3. A short longitudinal incision has been made through the anterior wall of the rectum at the site of the needle puncture and a stiff rubber tube inserted. The tube is held in place by means of one suture through the rectal wall. The end of the tube is left long and extends out through the anus.

Diagnosis. A pelvic abscess should always be suspected in any patient who, following operation, continues to run a septic course. Wound infection and other causes of fever, e.g., urinary tract infection, pneumonia, thrombophlebitis, etc., should, of course, be considered and excluded. Abscess formation in the cul-de-sac is always to be looked for in patients being treated conservatively for general peritonitis. Daily rectal examinations should be made. An inflammatory mass or infiltration is easily felt as a hard, firm, tender tumor mass bulging into the upper rectum anteriorly. It is usually felt at the tip of the examining finger. Subjective symptoms referable to the bladder, e.g., dysuria, urgency or frequency may be present. In the later

stages, diarrhea is not uncommon. Repeated digital examination of the rectum will enable the surgeon to follow changes in the size of the mass. Thus it may diminish, an indication of spontaneous resolution, or it may enlarge downward. Areas of softening indicative of suppuration should be sought for. Large cul-de-sac masses may arise out of the pelvis and then become palpable on abdominal examination. As suppuration occurs in a pelvic mass, the anal sphincter relaxes and the anus becomes patulous. The rectal mucosa becomes edematous and succulent and has a soft, thick, velvety feel. When these findings are encountered, surgical drainage is indicated. In general, our preference is for abdominal drainage and this should be performed in the extraperitoneal manner. Many surgeons, however, more or less routinely advise drainage by way of the rectum or vagina. In rectal puncture there is always the danger of injury to the great vessels of the pelvis. The bladder may also be injured during this procedure and Stafford and Sprong report a fatality due to this accident which was followed by a persistent rectovesical fistula and a fatal ascending infection of the urinary tract. In our opinion, rectal drainage should be reserved for those pelvic abscesses which are large and which point low down in the rectum, and in which a large area of softening can be demonstrated. When this is the case, the diagnosis is first confirmed by aspiration of the abscess with a large gauge needle. (Fig. 2.) The bladder having been emptied previously, the patient is placed in the lithotomy position. If pus is obtained on aspiration, drainage of the cavity is established by making a vertical incision in the anterior rectal wall at the site of the needle puncture, evacuating the pus and inserting drains. (Fig. 3.) In properly selected cases, this is a very simple technical procedure and provides an efficient form of drainage. Undrained cul-de-sac abscesses may spontaneously rupture into the rectum and thereby undergo spontaneous cure.

Left Lower Quadrant Abscesses. Abscesses in the left lower quadrant of the peritoneal cavity occasionally occur but are relatively uncommon. They are encountered more frequently as a postoperative complication than in patients undergoing conservative treatment for general peritonitis. Barrow and Ochsner call attention to the fact that abscesses in this locality are seen only in patients with a shallow pelvis and that they are most often found in children. They frequently are associated with an abscess which fills the pelvis and then extends upward and points to the left. The general signs and symptoms are those of any other secondary or residual abscess plus the localizing signs of tenderness, possible muscle spasm and skin edema, and a palpable mass in the left lower quadrant. The principles of treatment are similar. Here, again, it should be mentioned that many will subside spontaneously. If suppuration occurs, drainage will be necessary and is best performed through a small muscle splitting incision, taking care to avoid contamination of the main peritoneal cavity. As a rule, there is no great urgency for surgical intervention and, before proceeding with surgery, one should be certain that suppuration has taken place and ample time should be allowed to be certain that firm adhesions are present, in order that these protective barriers are not broken down in the process of drainage.

Subdiaphragmatic Abscess. Suppurative disease of the appendix is responsible for more instances of subphrenic abscess than any other single lesion. Ochsner and DeBakey[13] in their analysis of 3,533 cases of subphrenic abscess, collected from the world literature, found lesions of the appendix to be the etiological factor in 30.9 per cent, and in 25.3 per cent of their own seventy-five cases. In 15,000 collected cases of acute appendicitis, they calculate that subphrenic abscesses occurred as a complication in 0.9 per cent. They emphasize the point that many subphrenic infections do not progress to the stage of abscess and, also, the fact that due to

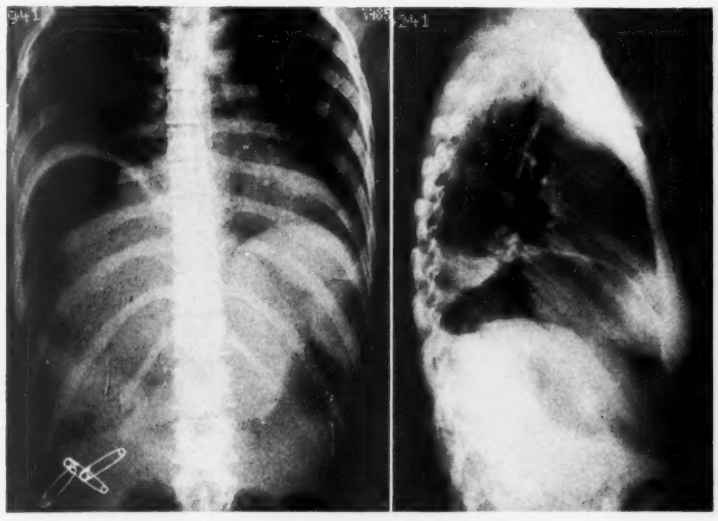
difficulties in diagnosis many subphrenic abscesses are never recognized. Faxon,[14] in a recent communication, reported 124 consecutive operative cases of subphrenic abscess at the Massachusetts General Hospital, and found the etiology to be a lesion of the appendix in thirty-eight, or 31 per cent. Wellman and Maddock,[15] in a review of fifty-two cases of subphrenic abscess, observed at the University of Michigan Hospital over a ten-year period, found that twenty-one, or 40.3 per cent, occurred as a complication of appendicitis.

The mechanism whereby infection may reach the subdiaphragmatic spaces from the somewhat distant right iliac fossa is of interest. The route most often described is that of direct extension along the right paracolic gutter. Overholt,[16] by experimental studies, has demonstrated the fact that a negative pressure is created in the upper abdomen by respiratory movements, and this would tend to cause septic material to be aspirated upward and into this region. Considerable attention has been given to the possibility of extension by way of the lymphatics by Munro,[17] Barnard,[18] and Truesdale.[19]

In order to appreciate fully the significance of subphrenic infections, a brief review of the anatomy of the region is necessary. Following the earlier descriptions by Martinet[20] and Barnard,[21] the term subphrenic region has come to denote that space in the upper abdomen bounded superiorly by the diaphragm and inferiorly by the transverse colon and mesocolon. Thus, subhepatic abscesses properly belong in this group. Detailed descriptions of the anatomy of the region are found in the excellent treatises by Ochsner,[22,23,24] Nather and Ochsner,[25] Ochsner and DeBakey, Ochsner and Graves,[26] and by Faxon.[27] The main subphrenic space is divided by the liver into a suprahepatic and an infrahepatic portion. Each of these compartments is in turn subdivided into three smaller spaces. Passing from the diaphragm to the liver, the falciform suspensory ligament divides the suprahepatic space into right and left spaces of approximately equal size. The right suprahepatic space is divided by the lateral extension of the cardinal ligament of the liver, i.e., the right lateral ligament into a large right anterior superior space and a smaller posterior superior space, leaving a single left superior space. The infrahepatic space is also divided into right and left halves by the round ligament and the ligament of the ductus venosus. While there is but one right inferior space, on the left side and separated by the stomach and gastrohepatic omentum are the left anterior inferior and the left posterior inferior spaces.

The right posterior superior space is the one most commonly involved in all subphrenic abscesses, and this is especially true in those infections of appendiceal origin. Thus Ochsner and DeBakey found in a review of 1,461 cases of subphrenic abscess that the right posterior superior space was involved in 33.7 per cent, and in 55.7 per cent of their own seventy cases. In Faxon's study, the right posterior superior or right inferior spaces were involved in 66 per cent of the cases in which the infection originated in the appendix. Subphrenic abscesses may be residual, as in cases of a resolving general peritonitis, or secondary when infection has extended from the appendix to the peritoneum but when general peritonitis has not occurred. With regard to diagnosis, the condition is suggested by continued evidence of sepsis (fever and leucocytosis) in a patient known to have had a local or general appendiceal peritonitis and in whom evidence of a suppurative process elsewhere in the abdomen, as well as extra-abdominal causes for fever have been excluded. Faxon stresses the following points in the history and examination: (1) tenderness over the twelfth rib or lower costal margin, sometimes best elicited by compression of the thorax, (2) findings indicative of a high, fixed diaphragm on the affected side, and (3) manifestations of diaphragmatic irritation, e.g., pain referred

to the shoulder or neck, hiccoughs and discomfort on deep respiration. The liver is often displaced downward.



A B

Fig. 4. A, roentgenogram in anteroposterior projection showing the appearance in a late case of subdiaphragmatic abscess. The right diaphragm is elevated and a large gas bubble is seen beneath the diaphragm. B, lateral projection in same case as A. The large collection of gas under the diaphragm is shown.

The roentgenological examination will assist materially in the diagnosis, although the roentgen findings are not pathognomonic until the late stages when a gas bubble and fluid level can be demonstrated. (Fig. 2.) However, in conjunction with consistent clinical findings, roentgen evidence of a high fixed diaphragm and an associated pleural effusion calls for a presumptive diagnosis of subphrenic abscess. (Figs. 3 and 4.) In order to be of the greatest assistance, particularly in the localization of the infection, the roentgenological study should include, in addition to the usual anteroposterior films, lateral views as well as fluoroscopic examination. (Fig. 5.) In the case of patients who continue to have unexplained fever following perforation of the appendix, it is our practice to have roentgen studies of the diaphragm every third day since, in such instances, the possibility of infection in the subphrenic spaces must always be borne in mind. It is to be remembered that at the time of laparotomy or abdominal paracentesis, air may be introduced into the abdominal cavity and since it may remain unabsorbed for as long as two weeks, its presence may confuse the picture. At the present time most authors condemn the practice of aspiration of the subphrenic spaces as a diagnostic procedure. It is an unreliable method, as failure to obtain pus does not exclude abscess. Moreover, the danger of contamination of uninvolved portions of the peritoneum, the pleura, or both, cannot be minimized. When, however, the question is one of differential diagnosis between subphrenic

Fig. 4. C, anteroposterior projection, with patient lying on left side. The large collection of air is shown and a definite fluid level is present. The irregular shadows in this film at the left are due to the injection of lipiodal into a sinus tract resulting from a previous unsuccessful attempt at drainage.

abscess and empyema, a carefully performed thoracentesis is not objectionable. Ochsner and DeBakey suggest that approximately 75 per cent of the cases of subphrenic infection will subside spontaneously under conservative measures, and suppuration demanding surgical drainage will occur in only 25 per cent. They believe that when the aforementioned findings suggesting subphrenic space infection fail to subside within a week, it is justifiable to assume the presence of an abscess and to proceed with surgery. In cases in which the diagnosis remains in doubt, exploratory operation is advised by them since, if performed extraserously, the risk is slight. The technical procedures for extraserous drainage are well described in the papers by Ochsner devoted to this matter. Undrained subphrenic abscesses may rupture through the diaphragm, with a resultant empyema, bronchopleural fistula, pneumonia or lung abscess.

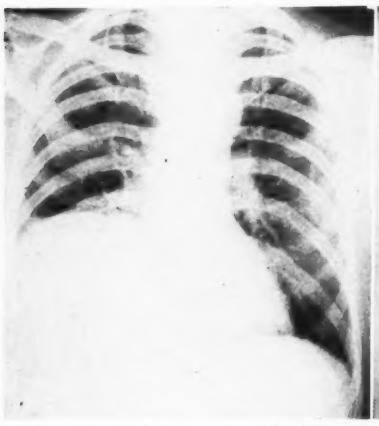


Fig. 5. Early roentgen findings in a case of subdiaphragmatic abscess. The right diaphragm is elevated and there is partial obliteration of the costophrenic sulcus due to pleural effusion. Fluoroscopy showed the diaphragm on this side to be immobile.

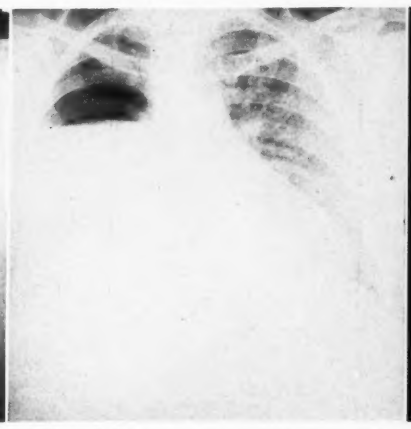


Fig. 6. Roentgenogram in case of right subphrenic abscess with an unusually extensive associated pleural effusion. In this case, repeated thoracenteses were performed before the correct diagnosis was made. It was after these were done that the fluid level appeared, due to the air introduced at the time of the taps. Cultures of the fluid were consistently negative.

Fig. 7. Roentgenogram in case of subdiaphragmatic abscess involving the right anterosuperior space. This shows the value of the lateral projection in the localization of the abscess.

Right-sided or Ileocecal Abscess. These local abscesses are relatively uncommon as postoperative complications, especially in cases where drainage has been employed. They are most likely to occur when, due to errors in judgment, drainage was omitted at the time of the original operation. In the early stages before a definite mass has developed, they may be confused with a wound infection. The symptoms and signs may be similar. In each there is continued fever, tachycardia, leucocytosis, pain, tenderness, swelling and induration in the neighborhood of the wound. Later, in the case of an ileocecal abscess, an intra-abdominal mass becomes recognizable. In either case, whether localized intraperitoneal infection or wound sepsis, the early treatment is conservative, i.e., hot wet dressings, physiological rest and chemotherapy. If an intra-abdominal abscess is suspected, the usual conservative measures for peritonitis should be employed in order to facilitate localization and to minimize the danger of spread of infection beyond the right iliac fossa. Under these conditions the mass becomes more localized and later may increase in size if suppuration occurs. Suppuration is disclosed by the presence of softening or possibly fluctuation, while the temperature chart reveals a "picket

fence" type of curve. When this takes place, surgical drainage is indicated. However, the operation should not be performed until one is certain that the surrounding adhesions are sufficiently strong so as not to be broken down easily, since contamination of the remainder of the peritoneal cavity must be avoided and every effort should be made to perform an extraperitoneal type of drainage operation. In cases of general peritonitis, treated conservatively by the Ochsner regimen, the right iliac fossa is a common site for localization of the infection and subsequent abscess formation. The diagnosis and treatment of these abscesses are essentially the same as in the case of those which occur postoperatively. It is important to note that the majority of abscesses encountered in the right lower quadrant occur not as a postoperative complication, but rather primarily following the slow perforation of an infected appendix. Far more common than the true abscesses, are the inflammatory masses or infiltrations in this region, and a distinction between these two lesions should be made. In the treatment of such inflammatory masses, whether true abscesses or inflammatory infiltrations, Lehman and Parker[28] advocate conservative therapy. During the years 1936 to 1937, 83.3 per cent of their abscess cases were treated along conservative lines and 65 per cent were carried through without operation. Under certain conditions they found it necessary to abandon conservative therapy and resort to surgical intervention. Such operative procedures are classed as "forced operations." They regard as indications for a forced operation during the course of conservative treatment, acute intestinal obstruction, or growth in the size of the abscess, with threatened perforation into the anterior abdominal wall or rectum along with an associated increase in the signs of sepsis. It has been pointed out by Wangensteen[29] that prolonged delay in draining large abscesses is attended with certain dangers, since such an abscess may rupture into the free peritoneal cavity, an accident which is almost invariably fatal, as the unprepared peritoneum is then suddenly flooded with septic material and an overwhelming infection of the peritoneum ensues. Again, such an undrained abscess may burrow into the mesentery and cause erosion of a large blood vessel, with disastrous results.

DISORDERS OF THE LIVER

Pylephlebitis and Liver Abscess. Fortunately, hepatic abscesses occur but rarely as a complication of the septic appendix, but constitute a most serious problem when present. The mode of spread of infection whereby suppurative appendicitis is responsible for hepatic abscesses is most often by way of the portal vein, i.e., following pylephlebitis. A less common cause is extension to the liver from a subphrenic space infection or abscess. Ochsner, DeBakey and Murray,[30] in a series of 575 cases of pyogenic abscess collected from the literature, found 197, or 34.2 per cent, to be secondary to appendiceal disease. In their own series of forty-seven cases of pyogenic liver abscess, five, or 10.6 per cent, were due to appendicitis, while in their personal series of 5,293 cases of acute appendicitis over a ten-year period, there were five instances of secondary liver abscess, an incidence of 0.094 per cent. The liver abscesses complicating acute appendicitis are usually multiple, and the right lobe is more commonly involved than the left. Occasionally, small abscesses may coalesce to form a large solitary abscess.

The usual clinical picture of pylephlebitis and liver abscesses complicating acute perforated appendicitis is characterized by high fever and chills, usually of abrupt onset. There is localized pain and tenderness in the region of the liver. The liver is enlarged. In the study of Ochsner, DeBakey and Murray, jaundice proved to be a less common finding than is generally supposed. These authors found jaundice to be present in only 36.4 per cent of their collected cases, and 24.7 per cent in their own series. When present, it was a late

development and they regard its presence as of grave prognostic import. Koster[31] emphasizes the importance of splenomegaly ascites and tenderness along the course of the portal vein as of diagnostic significance. The leucocyte count is often unusually high, with a proportional increase in the percentage of polymorphonuclear leucocytes. Roentgen studies are of value and show elevation and fixation of the diaphragm, particularly the right. In the series of Ochsner, DeBakey and Murray, a positive roentgen diagnosis was made in twenty-three of twenty-eight cases examined roentgenologically, or an incidence of 82.1 per cent. Koster suggests the use of thorium dioxide (thorotrast) in cases in which there is doubt of the diagnosis, and also for the purpose of determining the character and extent of the suppurative process in the liver. He administers the drug intravenously in doses of 25 cc. on three successive days, following which roentgenograms are taken. In normal persons the *x*-ray shadow of the liver is of uniform density. An abscess will appear as an area of lesser density, and in multiple abscesses the liver shadow will show a mottled appearance. Judging from the reported cases, this procedure seems to be of value as far as accuracy of diagnosis is concerned. However, sufficient evidence has not yet accumulated to determine whether or not thorotrast is entirely without danger. Most authors condemn exploratory puncture of the liver in doubtful cases. While the prognosis of pyogenic abscess of the liver is decidedly bad, this is particularly true in the case of multiple lesions, the type most often found in association with appendicitis. Thus in the series reported by Ochsner and his associates, the mortality for single abscesses was 37.5 per cent in contrast with a mortality of 95 per cent in the cases in which multiple abscesses were present.

Acute Hepatitis. Simeone and Stewart[32] report two cases of acute appendicitis with perforation and general peritonitis in which, during the postoperative period, bleeding due to hypothrombinemia occurred. This, they believe, was due to disturbed liver functions consequent upon an acute hepatitis which, in turn, was due to severe intraperitoneal sepsis. Since, under such circumstances, such bleeding may result in marked loss of blood and extension of infection, they recommend frequent plasma prothrombin determinations in cases of peritonitis. Since the hypothrombinemia is due to a depression of liver function, the administration of vitamin K is less effective than usual and, therefore, in addition to its use, repeated blood transfusions should be given.

ILEUS

Ileus, as a postoperative complication, may be of two types, (1) acute mechanical intestinal obstruction and (2) adynamic or paralytic ileus. A distinction should be made between these two types, although in some instances the two may co-exist.

VanBeuren[33] reports that of 130 patients with acute ileus operated on at the New York Presbyterian Hospital from 1932 to 1935, eight, or 6 per cent, occurred as complications of acute appendicitis. The incidence of late obstructions due to adhesions following operations for acute appendicitis would be considerably higher. VanBeuren also found that in 380 patients with acute appendicitis coupled with acute diffuse peritonitis operated on at the Presbyterian Hospital between 1916 and 1938, acute ileus developed as a postoperative complication in twenty-eight cases, or 7.4 per cent. This author reports a mortality of 56 per cent in a series of thirty-four cases of acute ileus complicating acute appendicitis with abscess or general peritonitis. In this group of cases, the mortality of the mechanical type of ileus was 47 per cent, whereas that of the paralytic type was 63 per cent.

Acute Mechanical Obstruction. Due to adhesive bands, a loop of bowel, usually the terminal ileum, may be caught and angulated so that a mechanical obstruction is produced in cases of localized or general

peritonitis. While these recent light adhesions are unlike the dense, firm adhesive bands seen in late adhesive obstructions, nevertheless they are entirely capable of causing complete mechanical blockage to the flow of the intestinal stream. This is made possible because of the fact that in peritonitis, and especially in patients having had an operation involving an unusual amount of trauma or manipulation, there is associated some degree of paralytic ileus. Due to the consequent loss of the normal propulsive power of the intestinal muscle, the coils become dilated with fluid and gas and a dangerous angulation at the point of fixation occurs. Great distention of the bowel interferes with its blood supply and, if not corrected, may lead to necrosis of the wall with perforation. Many of the postoperative obstructions, therefore, represent a combination of mechanical and adynamic ileus.

While the diagnosis of small intestinal obstruction due to adhesive bands but occurring weeks, months or years after operation is ordinarily easily made, the recognition of this type of obstruction which occurs during the postoperative period may be more difficult. Here, as in postoperative general peritonitis, the symptoms may be relatively mild and insidious. They often are overshadowed by persistence of the symptoms and signs of the original peritonitis. Thus abdominal pain and obstipation are difficult of evaluation. However, during the postoperative period, any increase in pain, especially if it is colicky and cramp-like in character, should be the cause for concern. Nausea and vomiting, if persistent and severe, should suggest mechanical or paralytic ileus. The presence of "fecal" vomiting is indicative of a low obstruction. Abdominal distention is due either to mechanical or adynamic ileus. In mechanical obstruction, auscultation reveals active peristalsis. This examination likewise is of less value than in those patients who are not convalescing from an abdominal operation and peritonitis. *X*-ray examination is of the greatest importance. The simple flat plate or scout film of the abdomen, taken with the patient in the supine position, is usually sufficient. The administration of barium by mouth is absolutely contraindicated and a very ill patient should not be subjected to complicated or prolonged examinations. Wangensteen[34] has pointed out that while gas is normally present throughout the gastrointestinal tract, it is visualized in the *x*-ray film of the abdomen only in the stomach and colon, except in the case of patients under three years of age. In the small intestine, while gas is present, it is so intimately mixed with fluid that it is not visualized under conditions of normal peristaltic activity. When stasis occurs due to mechanical or functional ileus, the fluid and gas separate and gas can be demonstrated. If ileus is present, dilated small bowel may be seen within a few hours following the onset, and subsequent films may demonstrate a progression of the process. Dilated small bowel is recognized by its central position, its feathery cross striations and the transverse position of the parallel coils. (Fig. 8.) The presence of many coils suggests a low obstruction, and any great degree of asymmetry suggests a mass as the cause of the displacement. (Fig. 9.) By means of the roentgenogram alone, it is impossible to distinguish between mechanical and adynamic ileus. Blood chemistry studies show an increase in the nonprotein nitrogen, a fall in the plasma chlorides and usually an increase in the carbon dioxide combining power. In these early postoperative adhesive obstructions, treatment by gastroduodenal suction after the method of Wangensteen[35] or, preferably, by the use of the "long" Miller-Abbott tube, is eminently successful. In this type of case, in the past, simple enterostomy usually effected a prompt cure. The decompression of the distended coils of bowel above the obstruction served to release the kink at the site of fixation and, with the resumption of peristaltic activity, this complication was corrected. At the present time, intestinal suction

achieves the same result by means of what might be termed as "internal enterostomy" and surgical intervention is rarely necessary. Occasionally, in low obstructions in which adequate decompression is impossible by means of duodenal suction and in which technical difficulties are encountered in the passage of the Miller-Abbott tube beyond the pylorus or the ligament of Treitz, enterostomy may be necessary in order to avoid further delay and, in the very ill patient, to avoid the exhausting effects of repeated fluoroscopic manipulations. Still less frequently, gastroenteric suction may fail to relieve the obstruction and reoperation becomes necessary in the event that it is due to some unrelated condition.

Fig. 8. "Scout film" of abdomen in case of small intestinal obstruction. This shows the parallel arrangement and transverse position of the intestinal coils and their central location in the roentgenogram. The fine cross-striations are characteristic of dilated small intestine.

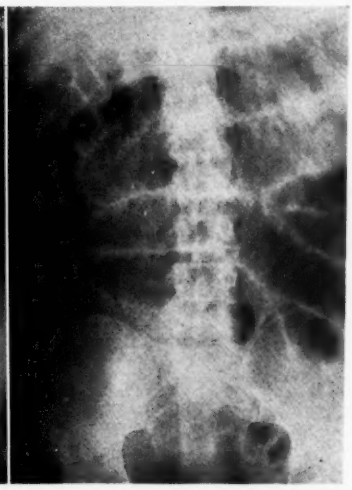


Fig. 9. "Scout film" of abdomen showing high grade obstruction of the small bowel with gaseous distention of the ileum and jejunum. There is an absence of bowel pattern in the right lower quadrant, suggesting the presence of a mass at that point.

Adynamic Ileus. Adynamic, paralytic or functional ileus is an accompaniment of peritonitis and may be regarded as a protective mechanism insofar as, with its occurrence, peristaltic activity ceases. Here, there is no single point of occlusion of the lumen of the bowel. Due to the loss of its contractile power, the intestine dilates and remains motionless. It becomes filled with its normal secretions and, since these are not moved onward, upper gastrointestinal stasis ensues. Due to reverse peristalsis, vomiting follows. Pain ordinarily is not severe or characteristic. The abdomen is distended and is silent on auscultation. Due to pressure on the diaphragm, respiration may be embarrassed. As already mentioned, mechanical and functional ileus may frequently co-exist.

The treatment of adynamic ileus is the treatment of the underlying peritonitis. The patient should be kept in the Fowler's position. Here, the use of duodenal suction by the method of Wangensteen is of the greatest value. Gastroduodenal suction serves to keep the upper portion of the digestive tract free of gas, swallowed air and retained secretions. Drugs such as eserine or pituitary derivatives are contraindicated because of the danger of spreading infection if violent peristalsis is stimulated. Moreover, they are inefficient in restoring the normal tonus and rhythmical contractions of the bowel.

Morphine, on the other hand, has a stimulating effect upon the intestinal musculature by increasing tone and rhythmic contractions, as shown by Orr.[36] Since it also minimizes the discomfort of the indwelling duodenal tube and the intravenous needles, it should be used freely. Plasma chlorides should be maintained at a normal level. Ochsner[37,38,39] recommends heat applied to the abdomen by means of body bakes. He believes that, due to the vasodilatation produced in the abdominal wall, there is a reciprocal vasoconstriction in the splanchnic areas which, in turn, has a beneficial effect upon distention. Distention may also be combated by oxygen therapy employing high concentrations of oxygen as recommended by Fine.[40] As the body defenses begin to overcome the infection, the tone and activity of the bowel will return to normal.

FECAL OR INTESTINAL FISTULA

Fecal fistula is an uncommon complication of operation for acute appendicitis, either with or without an associated peritonitis. In a series of ninety-four cases of intestinal fistula seen at the University of Michigan Hospital between the years of 1925 and 1934, and studied by Ransom and Coller,[41] thirty, or 32 per cent, developed following operations upon the appendix. Eight of these developed in the hospital following operation. In all of them the infection had extended beyond the appendix at the time of the original operation, and there was either a local abscess or a general peritonitis. These eight fecal fistulas occurred among 234 cases of acute appendicitis with abscess or general peritonitis, and which were treated by operation, an incidence of 3.4 per cent. The incidence of fistulas for the total number of operations for acute appendicitis with and without perforation (1,067) was only 0.8 per cent. In a series of 109 fecal fistulas observed in the Johns Hopkins Hospital over a period of forty years, Lewis and Penick[42] found that forty-nine, or 44.9 per cent followed appendicitis. Marshall and Lahey[43] found perforated appendicitis to be the etiological factor in ten of the thirty-seven cases of abdominal fistulas reported by them. These authors state that the most common cause of intestinal fistula is an infection which produces gangrene and perforation of the bowel and that, in their experience, cecal fistulas usually were the result of a direct extension of an infection of the appendix to the cecal wall, with a resultant sloughing of the wall. Fistulas involving the small intestine were more often the result of secondary operations for pelvic abscess or intestinal obstruction and were due to injury to the small bowel at the time of operation. Dixon and Deuterman[44] mention that a fecal fistula may follow drainage of an appendiceal abscess in cases in which the appendix is not removed at the original operation, since a fecalith in the remaining portion of the appendix may cause the development of such a fistula. Probably in some cases an unrecognized and unrelieved intestinal obstruction in the terminal ileum results in perforation of the bowel at the point of the obstruction, whereupon a fistula ensues.

Because it is believed by some surgeons that postoperative fistulas are due to improper treatment of the appendiceal stump, there has been much discussion in the literature regarding the merits and disadvantages of invagination of the stump at the time of appendectomy. Some authors maintain that such a technic is responsible for a high incidence of fecal fistulas, whereas it is contended that simple ligation without invagination reduces this incidence. It has been our practice to invert the stump with a purse-string suture of catgut or silk, or by means of three mattress sutures of silk whenever the condition of the cecal wall will permit this procedure. We have seen no instance of fecal fistulas which we could attribute to invagination of the appendiceal stump, and similar experiences are recorded by Lewis and Penick and by Dixon and Deuterman. Some fistulas are caused by the use of improper drainage materials. Fortunately,

glass and hard rubber drains, once so commonly used, have been largely superseded by drains of soft rubber, guttapercha, or drains of the cigarette variety. A heavy gauze drain allowed to remain in contact with a suture line is apt to provoke leakage; hence, when cigarette drains are used, the gauze should not project beyond the end of the Penrose tubing. The tip of the drain may be placed near but not against the suture line. Moreover, when feasible, it is well to interpose the omentum between the suture line and the drain.

In the forty-nine cases of fistulas following appendicitis reported by Lewis and Penick, the opening was in the cecum in twenty-one, or 72.4 per cent, in the ileum in four, or 13.7 per cent, and in the appendix in a similar number of cases. In the remainder of their cases, the point of communication could not be determined. In the thirty fistulas associated with appendicitis studied by Ransom and Coller, the opening was in the terminal ileum in thirteen, in the cecum in four, in both cecum and ileum in two, in the sigmoid in one, and was undetermined in ten cases.

The diagnosis of an intestinal fistula ordinarily is not difficult. In the case of most cecal fistulas, the discharge is semisolid and is not especially irritating to the skin. The fistulas higher in the small intestine are associated with an irritating discharge and, due to the fluid character of the contents, considerable quantities of fluid, electrolytes and food substances may be lost. Occasionally, it may be difficult to distinguish between a fecal fistula and a colon bacillus infection of the wound or in a hematoma. Dixon mentions the importance of the history of the passage of gas in such cases as indicative of fistula and, in doubtful cases, suggests the use of a dye such as carmine red by mouth. Roentgen studies following injection of the fistulous opening with some radio-opaque material is often of value in localization. Injection in conjunction with a barium enema often gives additional information.

The fistulas associated with appendicitis tend to heal spontaneously. This is particularly true of the cecal fistulas. When healing fails to occur in these cases there is usually an outgrowth of the cecal mucous membrane so that it becomes continuous with the skin, producing the direct or lip type of fistula. In such cases, surgical closure is necessary. The ileal fistulas are more apt to be complete and are less likely to heal spontaneously than are those in the cecum. Here, the added complications of spur formation, herniation, or obstruction distal to the fistula are mechanical factors which serve to maintain the fistulous opening and they must be recognized and corrected by the proper surgical procedures. As a rule, in the fistulas associated with appendicitis, conservative treatment should be given a prolonged trial before resorting to operative closure. Even though a surgical repair may be necessary at a later date, a successful result is much more likely to be obtained when the local inflammatory reaction has been given a chance to subside before operation is attempted.

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JAUNDICE

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WHEN a case of jaundice is encountered, certain questions are at once evident: Is the jaundice attributable to hemolytic, obstructive or hepatogenous processes? Is a combination of causes present? Will surgical intervention be necessary for relief, and if so, what is the opportune time for operation and can the risk of such a procedure be reduced by preoperative medical measures? Are any special procedures advisable during the postoperative course? In many instances, these questions can be answered readily, but in others, to find the answers requires considerable search. As in the treatment of other diseases, experience adds measurably to the facility with which the internist arrives at his conclusions. No matter how extensive a physician's clinical experience may be, however, cases will be encountered in which a definite preoperative diagnosis cannot be made and in which the answer may have to await exploratory operation or examination at necropsy.

In elucidation of the problems of jaundice, it is necessary to have a working classification of the various causative factors. (Table 1.) Obstructive jaundice most commonly is due to stones in the biliary tract, cicatricial strictures of the common duct, malignant processes involving the head of the pancreas or ampulla of Vater, or metastatic lesions about the common bile duct or in the liver. Hepatogenous jaundice may be caused by a large variety of lesions, the most common of which are the various forms of hepatitis of endogenous, exogenous or idiopathic origin, cirrhosis and infections, such as catarrhal jaundice, Weil's disease and suppurative cholangitis. The rare case of constitutional hyperbilirubinemia also is probably of hepatic origin, such as demonstrated in Case 1. The hemolytic group includes pernicious anemia and congenital or acquired hemolytic icterus.

TABLE 1
CLASSIFICATION OF JAUNDICE

I. Obstructive jaundice
  A. Choledocholithiasis
  B. Cicatricial stricture of the common bile duct
  C. Malignant obstruction
    1. Carcinoma of the head of the pancreas
    2. Carcinoma of the papilla of Vater
    3. Carcinoma of the gallbladder and bile ducts
    4. Metastatic malignant lesions involving the liver, bile ducts or periportal lymph-nodes
  D. Acute or subacute pancreatitis
II. Hepatogenous jaundice
  A. Circulatory disease
    1. Chronic passive congestion
    2. Thrombosis of portal or hepatic vein
  B. Suppurative infections
    1. Cholangitis
    2. Portal pylephlebitis
    3. Hepatic abscess
  C. Nonsuppurative infections
    1. Epidemic catarrhal jaundice
    2. Weil's disease
    3. Yellow fever
    4. Malaria
    5. Syphilis
    6. Typhoid
    7. Pneumonia
    8. Influenza, etc.
  D. Toxic degenerations
    1. Exogenous—arsenic, cinchophen, alcohol, chloroform, etc.
    2. Endogenous—hyperthyroidism, toxemias of pregnancy, etc.
    3. Idiopathic—yellow atrophy
  E. Cirrhosis
  F. Constitutional hyperbilirubinemia
III. Hemolytic jaundice
  A. Hemolytic icterus—congenital or acquired
  B. Pernicious anemia
  C. Transfusions of incompatible blood
  D. Toxins—phenylhydrazine, etc.
  E. Infections—malaria, bacterial endocarditis as examples
  F. Miscellaneous hemolytic processes

An accurate and detailed history of attacks and symptoms is also necessary in elucidation of the problem. In a large

majority of cases a complete and accurate history will permit a satisfactory classification of the type of jaundice present. Confirmatory evidence and more detailed information can be obtained from physical examination and the various laboratory procedures, including some of the many tests for hepatic function.

HISTORY

Stone in the common bile duct is the most frequent cause of jaundice that is amenable to surgical operation. The diagnosis in such cases is best established by the history. Since this is true I first shall describe the symptoms which stone in the common duct may produce. I then shall change the point of view and consider certain symptoms often mentioned by patients who have jaundice and suggest the underlying disease.

Stone in the Common Duct. The classical story of chronic indigestion of gaseous type, with qualitative and quantitative relation to food, together with the occurrence of biliary colic, is well known. Many variations, however, are common. Chronic indigestion may be absent. Colic may be absent or atypical in character or location. The patient may complain of short spells of indigestion, commonly represented by a sensation of fullness, pressure or aching in the upper portion of the abdomen. At times, relief is obtained by induced belching, vomiting, or bowel movements. These attacks may occur at any time of the day or night. Nocturnal awakening suggests organic disease. The attacks may last from a few minutes to an hour or more and tend to occur with increasing frequency and to persist longer. There may not be any residual soreness. Eventually, a physician's care is required. As time progresses, other symptoms or signs may be manifest, such as slight fever, mild chills, residual soreness in the right upper quadrant of the abdomen and even mild jaundice of transitory nature. The alert physician recognizes the significance of these signs and symptoms at once. Some patients, however, delay seeking medical advice until their symptoms are more severe and associated with marked decline in their general health. Patients for whom diagnosis of stone in the common duct often is difficult from the history alone are those of advanced years who always have enjoyed good health. Their tolerance for pain is high; they tend to minimize the significance of indigestion and find it difficult to give an adequate account of their symptoms. Their age and previous good health always arouse suspicion of possible malignant disease when any digestive symptoms develop.

Colic Followed by Jaundice. In general, however, the history of attacks of colicky pain in the upper portion of the abdomen followed by jaundice means the presence of stone in the common duct. Such definite attacks frequently are associated with chills and fever. Jaundice after such attacks varies in duration and degree; commonly, it is relatively mild (the value for serum bilirubin averages 10 mg. per 100 cc.) and tends to clear in from one to two weeks. In other instances, it is deeper and persists indefinitely, although it may fluctuate considerably. Exacerbations of pain often precede increase in jaundice. With the onset of jaundice, concomitant discoloration of the urine and lightening of the color of the feces occur, although many patients do not observe these changes until their attention has been called to them. Such changes aid little in determining the cause of the type of jaundice present.

Although colic followed by jaundice usually means stone in the common bile duct, a similar history occasionally is encountered in cirrhosis of the liver, at the onset of jaundice as a result of carcinoma of the head of the pancreas and of acute pancreatitis. Cases II and III are illustrative of such conditions.

Jaundice without Pain. Onset of jaundice not associated with pain is attributable to a wider variety of causes and offers a greater problem in diagnosis, especially if bile can be demonstrated entering the

intestine. The physician should be particularly careful to inquire concerning the ingestion of hepatotoxic drugs, the presence of epidemic jaundice, the history of preceding infection of the respiratory or gastrointestinal tract and any history of preceding, mild attacks of jaundice. Jaundice of painless onset among young patients is most frequently hepatogenous; among older patients, it most frequently is attributable to malignant lesions obstructing the biliary tract, especially if the jaundice is intense, persistent and associated with marked pruritus. Absence of bile in the intestine and a palpable, distended gallbladder usually establish the diagnosis in this type of jaundice. In such cases, there may be a preceding history of a few months' duration characterized by indefinite discomfort in the upper portion of the abdomen, impairment of appetite, loss of weight, and, at times, mild diarrhea. The diarrhea consists of occasional stools which are bulky, loose and light-colored because of excess fat. Occasionally, jaundice occurring without pain is attributable to stone in the common bile duct. If expectant management is employed, the occurrence of pain followed by increase or decrease of icterus may clarify the situation. In some instances, physical examination and results of various laboratory procedures may elucidate the nature of difficult problems. Again, it may be necessary to undertake exploration before a satisfactory explanation is found.

Pruritus. Pruritus is a common accompaniment of obstructive jaundice. It is, however, often absent or minimal in degree in cases of jaundice attributable to stone in the common bile duct, especially if jaundice is mild. It more frequently is present and commonly is more intense in the deep jaundice that accompanies malignant obstructions of the bile ducts. Pruritus is absent in hemolytic jaundice unless some complicating factor, such as choledocholithiasis or hepatic cirrhosis, is present. A variable number of patients who have hepatogenous jaundice suffer from pruritus. Commonly, cases are encountered in which severe degenerative lesions of the liver are associated with intense jaundice, such as acute yellow atrophy, without pruritus. Pruritus is infrequently a symptom of atrophic cirrhosis. Nevertheless, pruritus of severe degree, persisting over long periods and associated with mild, slowly deepening jaundice and melanosis, usually is indicative of a certain type of primary hepatic disease. Confirmatory evidence usually can be obtained by physical examination, tests for hepatic function and subsequent course of the patient.

Diarrhea and Steatorrhea. Diarrhea is an uncommon symptom in jaundiced conditions, although in cases in which it occurs it appears to be due to insufficient bile in the intestine to permit adequate absorption of ingested fats. In cases of obstruction of the pancreatic duct, steatorrhea may be present for some weeks or months before the malignant lesion producing obstruction involves the bile duct and causes jaundice.

Edema, Ascites and Hematemesis. A history of edema and ascites not attributable to cardiac or renal disease usually means extensive primary or malignant disease of the liver, with obstruction of the portal system and other changes. However, in a few long-standing and neglected cases of stone in the common bile duct with secondary biliary cirrhosis, such symptoms may be present. Hematemesis usually has a similar significance, although in an occasional case it may be due to an associated lesion, such as gastroduodenal ulceration or malignant disease.

DIAGNOSIS IN CASES IN WHICH OPERATION HAS BEEN PERFORMED PREVIOUSLY

For the patient who has had previous surgical operation on the gallbladder or bile ducts and whose presenting symptom is jaundice, accurate diagnosis is much more difficult. A knowledge of the symptoms that led to the operation, the operative findings and procedures is almost

imperative; this information should be obtained from the patient or surgeon. Many combinations of symptoms may be encountered in such cases. The simplest would be a preoperative history of gallstones and jaundice and acute inflammation of the gallbladder found at operation, with establishment of drainage the only surgical procedure performed. In these cases, the surgical treatment obviously is incomplete. When circumstances permit, a second operation with opening of the common bile duct is indicated. Stone in the common bile duct usually is found, although in some instances edema of the biliary tract and pancreas attributable to the infection may cause jaundice. However, exploration of the common bile duct usually is necessary. In other instances of cholecystic disease, the gallbladder has been removed; it may or may not have contained stones and convalescence may have been normal. Sooner or later, colic associated with fever and, at times, chills and jaundice develop. This situation most frequently is attributable to stone in the common bile duct. If colic develops within a short time after operation and if some jaundice has been present before operation, an overlooked stone is the most likely cause of the trouble. If some years have passed before the onset of symptoms, a stone of postoperative origin is probably the cause of the trouble. Postoperative colic may occur for months or years before jaundice develops; in such cases, exploratory operation is indicated.

Stricture of a cicatricial nature is the next most common cause of postoperative jaundice. Various combinations of symptoms have been described in this condition. In the usual case, the course is stormy after cholecystectomy, characterized by development of jaundice within two or three days postoperatively, with pain, fever and spontaneous development of an external biliary fistula. The fistula tends to persist. With development of the fistula, the jaundice may clear and pain and fever subside. As time progresses, however, the fistula tends to close and the pain and jaundice recur. Re-establishment of the fistula relieves the symptoms. In complete stricture, persistent jaundice and fistula are not unusual. In incomplete stricture, the fistula may close permanently. Jaundice may or may not have disappeared entirely but soon recurs or deepens in association with attacks of colic-like pains, chills and fever. These attacks increase in frequency, the jaundice tends to become permanent and all bile may disappear from the stool. The liver and spleen frequently become enlarged. In other cases, convalescence after cholecystectomy is much milder. Attacks of biliary obstruction, characterized by pain, chills, fever and jaundice develop after the wound has healed, increase in frequency and eventually reach a stage of permanent jaundice. I encountered a case in which these symptoms began two years after uncomplicated cholecystectomy was performed.

Cholangitis is a third factor in the cause of jaundice and colic after cholecystectomy. Its pathogenesis and mechanism are not well understood. Usually, stone or partial stricture is suspected until the common bile duct is explored. Even then, a small stone may be overlooked. Prolonged drainage of the common bile duct may not give the expected relief.

Biliary fistula persisting after cholecystostomy usually is attributable to residues of cholecystic disease, stone or obstructive lesions of inflammatory or neoplastic nature of the common bile duct. Further surgical procedures usually are required. From time to time, as the fistula closes, pain and jaundice may develop. If pancreatic enlargement was found at operation and cholecystostomy was performed merely to relieve the jaundice and pruritus, or as a phase of a procedure in two stages, in which subsequent anastomosis between the gallbladder and stomach or duodenum is planned, the external flow of bile usually is profuse and none enters the intestine. Dehydration and other nutritional changes are common in such instances. Persistent

fistula after cholecystectomy usually is due to an overlooked stone in the common bile duct or stricture. Jaundice may or may not be present. If drainage of the common bile duct has been established and external flow of bile continues after removal of the tube, some obstructive lesion in the lower portion of the common bile duct usually is present, such as overlooked stone, stricture, pancreatitis or neoplasm of the ampulla of Vater or pancreas.

PHYSICAL EXAMINATION

On examination of jaundiced patients, yellow discoloration of the skin is first noted. An impression of the degree or intensity of jaundice thus is obtained, although in cases of deep icterus the degree of bilirubinemia seldom can be gauged accurately. The tint of pigmentation also is noted. A lemon-yellow tint of relatively mild degree suggests the presence of a hemolytic process. An intense orange is indicative, although not always, of hepatogenous jaundice, and a green tint suggests obstructive jaundice of considerable duration. Pigmentation, aside from that attributable to retention of bile often is encountered in cases of obstructive or hepatogenous jaundice of long standing. This usually is brownish and is attributable to melanosis. Scratches may be numerous if pruritus is present.

On abdominal examination, the size, consistency and contour of the liver are determined and the presence of tenderness or rigidity is noted. Palpable enlargement of the gallbladder is searched for, and its size, contour, movability, consistency and tenderness are noted. Enlargement of the gallbladder usually is attributable to empyema, obstruction of the cystic duct and obstructions at the ampulla of Vater. Discernment of an enlarged gallbladder often is difficult, especially if the abdominal walls are tense and thick. Among obese patients, it seldom can be palpated. At times it is difficult to differentiate a distended gallbladder from an irregular margin of the liver.

Other points worthy of note include the presence of abdominal scars, fistulas or sinuses, splenic enlargement, distended veins, other abdominal masses and ascites. In the general examination, special note is made of enlarged lymph-nodes, possible metastatic nodules, including those in the rectum; pallor, cyanosis, purpura or other evidence of bleeding; clubbing of the fingers; spider angiomas; distended veins; edema; bile tophi; loss in weight and temperature.

Patency of the Bile Ducts. The next step in a study of the jaundiced patient is a determination of patency of the bile ducts; in other words, to determine whether bile is entering the intestine and in what quantity. Chemical examination of the feces for bile and use of duodenal intubation usually establish this satisfactorily. It should be noted that at times chemical tests for bile may be negative, although variable amounts of bile can be demonstrated entering the intestine by duodenal intubation. In choledocholithiasis, bile usually can be demonstrated entering the intestine, although occasionally a stone may be impacted firmly in the region of the ampulla of Vater, thus completely obstructing the passage of bile. In carcinoma of the ampulla or pancreas, "dry drainages" usually are encountered, especially in the later stages. In earlier stages the jaundice may even clear to a great extent for a short time; frequently, in such cases, blood may be obtained on duodenal aspiration. In performance of duodenal drainage, adequate control of the method is essential in order to be certain that the duodenum has been entered.

LABORATORY EXAMINATION

Roentgenoscopic Examination. Roentgenoscopic examination does not aid greatly in the diagnosis in cases of jaundice. Cholecystography is of no value because of hepatic disease or obstruction of the biliary tract. Occasionally, roentgenograms of the region of the gallbladder will demonstrate stones, but rarely is a shadow seen that

suggests stone in the common bile duct in cases in which cholecystectomy has been performed. Roentgenoscopic examination of the gastrointestinal tract is valuable in excluding primary malignant lesions that may have metastasized to the liver. Occasionally, widening of the duodenal loop may indicate enlargement of the head of the pancreas. Choledochograms are valuable in studying biliary fistulas and, especially, patency of the common bile duct if drainage tubes are in place.

Tests for Hepatic Function. The number of tests for hepatic function are almost legion. Too frequently, more information is anticipated from these tests than their originators indicated. Again, many tests are used and the results are overemphasized or misinterpreted; often, too, the data obtained from the history and physical examination are ignored. It generally is recognized that there is no one test that gives an entire picture of the condition of the liver and that no test will distinguish between hepatogenous and obstructive jaundice. Furthermore, employment of any test depends on its simplicity and accuracy. At the Mayo Clinic, a great many of the various tests for hepatic function have been employed, but, after experimental clinical trials, their use has been discontinued. Some of the tests that are still utilized will be considered herein:

The van den Bergh Reaction. Qualitatively, the van den Bergh reaction is always direct in cases of well developed obstructive and hepatogenous icterus. The reaction, however, does not aid in distinguishing between these types. Theoretically, the reaction is indirect in cases of hemolytic jaundice, but in cases of more marked icterus in which the value for serum bilirubin is from 6 to 10 mg. per 100 cc., a direct reaction commonly is encountered. In certain of the cases of hemolytic jaundice, however, direct reaction indicates the presence of a superimposed obstructive element, such as stone in the common bile duct or early hepatic damage.

Quantitatively, the reaction is valuable in determining the degree of jaundice and in following fluctuations in its intensity from time to time. The degree of bilirubinemia gives some information in regard to the condition of the liver. For example, in calculous obstruction of the common bile duct, the level of serum bilirubin is commonly from 8 to 12 mg. per 100 cc. If obstruction is complete, this level may be increased to 18 to 20 mg. However, levels higher than this indicate definite hepatic damage, apparently in direct proportion to the degree of retention. In carcinomatous obstruction, which may be more or less complete, levels of from 15 to 20 mg. are usual. Levels of 25 mg. or more have a significance similar to high levels present in cases of choledocholithiasis; levels as high as 50 mg. have been encountered. In hepatogenous jaundice, high levels have a similar significance; levels as high as from 15 to 20 mg. are not unusual and values of from 30 to 40 mg. indicate severe hepatic damage.

The van den Bergh reaction also is valuable in excluding evidence of hepatic disease in such conditions as carotenemia, pigmentation attributable to atabrine and in demonstrating biliary or hepatic disease in borderline cases, such as early hepatitis, cirrhosis or toxic conditions in which gross icterus is not present. The test also is valuable after attacks of abdominal pain that lack distinguishing characteristics, demonstrating in such instances the presence of a latent jaundice.

The Icterus Index. This is a simple method of following the course of jaundiced patients, but it lacks some of the advantages of the van den Bergh reaction, such as distinguishing hemolytic jaundice, familial hyperbilirubinemia, latent obstructive or hepatogenous jaundice and carotenemia.

The Bromsulfalein Test. This test is seldom of value in cases of jaundice, except in determination of hepatic function in hemolytic or latent jaundice. Its chief importance is in a study of hepatic function in the absence of jaundice.

Determination of Blood Cholesterol and Cholesterol Esters. Increased values for blood cholesterol and cholesterol esters in obstructive jaundice and reduction of these values in association with degenerative hepatic lesions commonly are encountered. A marked lowering of the levels often has prognostic significance. In many cases in both types, however, fairly normal values may be obtained. Occasionally, marked elevation of cholesterol and cholesterol esters occurs in chronic degenerative conditions, as demonstrated in Case IV.

Synthesis of Hippuric Acid and Galactose Tolerance Tests. These tests give some information concerning the function of the liver, but they do not aid greatly in distinguishing between obstructive and hepatogenous icterus. In many cases of each type, values may be normal. Again, synthesis of hippuric acid in obstructive jaundice may be decreased and excretion of galactose may be abnormally high, indicating a rather severe, associated hepatic disturbance in addition to the obstructive element. Conversely, abnormal results of these tests do not mean that an obstructive element is not present as a factor in producing jaundice.

Prothrombin Clotting Time. The prothrombin clotting time should always be estimated in cases of jaundice, both before and after administration of vitamin K and it should be estimated frequently in the course of convalescence of patients who have been treated surgically. A normal clotting time does not exclude the necessity of administering vitamin K, at least preoperatively and, for a period, postoperatively. Delay in the prothrombin clotting time demands delay in surgical procedures and indicates the urgent need of vitamin K. Failure to obtain reduction of the prothrombin clotting time from adequate administration of vitamin K usually indicates severe hepatic disease, even if the jaundice is primarily of obstructive origin.

Other Studies on Blood. Other studies of the blood occasionally give some definite information of the seriousness of disturbed physiologic processes in jaundice and in postoperative study of cases in which progress is not satisfactory. Included in these are estimations of urea, sugar, carbon dioxide combining power, chloride, protein, calcium and amino acids. In cases of severe jaundice, the kidneys frequently are involved and elevation of urea occurs. In certain cases of primary hepatic disease, lowered values for urea and sugar are encountered. If an external biliary fistula or drainage tube is present after operation, disturbances of the content of chloride and carbon dioxide combining power are at times encountered, indicating dehydration or acidosis. These changes occur most frequently if choleresis is manifest. The choleresis is a further indication of disturbance of hepatic function of considerable degree. These changes indicate that replacement or other treatment is necessary; and at times, they may show that it is necessary to apply a clamp or remove the drainage tube.

In some cases, especially cases of choleresis, a marked lowering of the cholesterol and bile salts in the excreted bile is present and indicates a serious prognosis. Occasionally, a patient who has chronic hepatic disease, including certain cases of hepatic disease of obstructive origin, either with or without external fistulas, may manifest marked osteoporosis with spontaneous fractures. The level of calcium in the blood usually is lowered. Lowered levels of serum proteins, especially albumin, are encountered commonly in primary hepatic disease and in cases in which the postoperative course is long and serious and the intake of food has been inadequate and hemorrhage or infection has occurred.

COMMENT

Disturbances of hepatic function as determined by one or more of the tests mentioned have definite clinical significance, although this varies with the degree of abnormalities. In all instances, however, the results of the tests should be subservient to sound clinical appraisal in

each case. Intensive preoperative treatment is indicated in those cases in which disturbances of hepatic function are present and surgical procedures are planned. Exploration should be deferred until all possible improvement has occurred and, when undertaken, adequate follow-up treatment is indicated. If satisfactory progress has not been made preoperatively, the operation may of necessity have to be proceeded with, in spite of the increased risk of such an undertaking. In the nonsurgical case, the results of these tests often serve as a guide in the treatment and progress of the patient. The following report of cases encountered at the Mayo Clinic illustrates some of the problems of diagnosis, the methods of using laboratory procedures and their significance.

CASE REPORTS

Case I. A woman, aged twenty-four years, had noted slight scleral icterus, slight indigestion and nausea after ingestion of fatty foods for six months prior to registration. The family history in regard to the blood, liver and spleen was negative. Pruritus was absent. The icteric index ranged from 12 to 25. The liver and spleen were palpable, but this may have been because of extensive scoliosis, causing displacement of the organs. On laboratory investigation, the value for serum bilirubin was 4.2 mg. per 100 cc.; the van den Bergh reaction was indirect. The cholecystogram disclosed a normally functioning gallbladder. The number of leukocytes and erythrocytes, differential count and morphologic elements of the blood were normal; fragility of erythrocytes was normal and sedimentation rate (Westergren method) was 6 mm. per hour. Retention of bromsulfalein was normal. The value of cholesterol was 196 mg. and that of cholesterol esters 120 mg., each per 100 cc. of plasma.

Comment. The only positive finding in this case was the hyperbilirubinemia. Investigation of the blood, liver and gallbladder failed to demonstrate any disease process. The hyperbilirubinemia in this case was considered attributable to constitutional hepatic dysfunction. Investigation in such cases is largely by means of laboratory procedures.

Case II. A man, aged forty-one years, had an attack of moderately severe colic in the right upper portion of the abdomen and vomiting one month prior to registration. Thirty-six hours after this attack jaundice, which deepened and persisted, pruritus, acholic stools and loss of twenty pounds (9.1 kg.) occurred. For two years he had suffered from mild indigestion. Examination revealed deep jaundice and slight enlargement of the liver. The value of serum bilirubin was 15.4 mg. per 100 cc.; the van den Bergh reaction was direct. At operation, a large, tense gallbladder, dilated common bile duct and tumor of the head of the pancreas were found. Cholecystogastrostomy was performed. The subsequent course confirmed the diagnosis of carcinoma of the pancreas.

Comment. The preceding indigestion and colic, followed by jaundice and absence of palpable gallbladder, were suggestive of gallstones. None were found. In carcinoma of the pancreas, the onset of jaundice may be associated with colic, thus simulating stone in the common duct. In some cases of carcinoma of the pancreas, stones occasionally are encountered in the gallbladder or common bile duct and may or may not have given rise to atypical symptoms.

Case III. A man, aged forty-six years, who had used alcohol moderately, had suffered from gaseous indigestion and attacks of pain in the right upper quadrant of the abdomen of varying severity and increasing frequency for fifteen years. These attacks had frequently been associated with chills and fever. For twenty years he had noted increasing dyspnea on exertion and for two weeks, dependent edema.

On admission the patient was suffering from severe pain. The temperature was 104°F. Moderate jaundice, tenderness in the right upper quadrant of the abdomen and edema of the lower extremities were present. Laboratory investigation revealed 27,400 leukocytes per cubic millimeter of blood, marked macrocytosis and a value for serum bilirubin of 6.2 mg. per 100 cc.; the van den Bergh reaction was direct. The value of cholesterol was 214 mg., and that of cholesterol esters 103 mg., each per 100 cc. of plasma. The value of total protein was

5.5 gm. per 100 cc. of serum. The albumin-globulin ratio was 1:1.6; excretion of hippuric acid was 0.37 Gm. Operation revealed a small, markedly hobnailed liver and some ascites. Gallstones were absent. Necropsy revealed cirrhosis of the liver.

Comment. The clinical picture indicated choledocholithiasis so strongly that exploration seemed indicated in spite of evidence of seriously impaired hepatic function, as indicated by edema, low value for total protein, decreased synthesis of hippuric acid and macrocytosis. Dyspnea, in the absence of cardiac, renal and pulmonary disease, and edema were clinical symptoms indicating chronic hepatic disease. Primary hepatic disease may thus mimic the syndrome of cholecystic disease and choledocholithiasis. The similarity is usually so striking that exploration cannot safely be avoided.

Case IV. The patient, a man aged forty-three years, presented a history of biliary colic of three years' duration. The last attack had occurred ten days prior to registration and was followed by deep, persisting jaundice. He had lost thirty pounds (13.6 kg.) in four months.

Examination revealed deep icterus and slight tenderness in the right upper quadrant of the abdomen. The value for serum bilirubin was 19.7 mg. per 100 cc.; the van den Bergh reaction was direct. Small amounts of bile were entering the duodenum; result of the galactose tolerance test was normal and the value for blood cholesterol was 340 mg. per 100 cc. At operation, the gallbladder was distended, thick-walled and contained white bile and pus, but no stones. A stone, 1.2 cm. in diameter, was removed from the lower end of the dilated common bile duct and a T-tube inserted for drainage. Marked hepatitis was present. Postoperatively, from 750 to 1,000 cc. of bile drained daily. A slight rise in the level of serum bilirubin occurred, followed by a slow decline. On the fifth day drowsiness was noted. These abnormalities were corrected by means of solutions of glucose administered intravenously.

Comment. The history in this case seemed to indicate choledocholithiasis. The presence of deep jaundice with incomplete obstruction of the common bile duct indicated hepatic damage in addition to the obstructive element. This was confirmed by appearance of the liver at operation and the presence of moderate choleresis, drowsiness, rise in the level of serum bilirubin and its slow decline postoperatively. The high level of serum bilirubin was the only evidence of hepatic damage as indicated by the laboratory procedures used preoperatively.

Case V. A woman, aged thirty-six years, complained of pruritus and spells of indigestion, anorexia and mild pain in the upper portion of the abdomen for two years. For a year the stools had been lighter and the urine darker than normal. Scleral icterus had been noted for three months. The patient had lost twenty pounds (9.1 kg.). Colic, chills or fever had not been experienced.

Examination revealed mild icterus, melanosis, marked excoriations of the skin and slight enlargement of the liver and spleen. Xanthelasma, edema, ascites or varicosities of the abdomen and esophagus were not demonstrated. The value for hemoglobin was 11.6 Gm. per 100 cc. of blood; erythrocytes numbered 4,160,000 per cubic millimeter of blood; macrocytosis was present and the value of serum bilirubin was 9.8 mg. per 100 cc.; the van den Bergh reaction was direct. Cholecystograms revealed gallstones. Excretion of hippuric acid was 3.7 Gm. Values were as follows: cholesterol 1,010 mg., cholesterol esters 694 mg., each per 100 cc. of plasma.

The diagnosis was primary hepatic disease associated with stones in the gallbladder. At operation, the liver was increased in size and firmer in consistency than normal. The gallbladder was opened, stones were removed and drainage was established. The common bile duct was normal in size, but was opened, probed and a T-tube inserted for drainage. During convalescence, 150 to 250 cc. of bile drained daily through the T-tube; one specimen contained 1,000 mg. of bile salts per 100 cc. The pruritus was relieved temporarily but recurred later. The jaundice remained stationary. The level of fats in the blood dropped moderately.

Comment. Some of the symptoms probably were attributable to gallstones. Absence of colic, chills, fever or fluctuations

in the jaundice and the long-standing, pre-icteric pruritus, together with enlargement of the liver and spleen, indicated primary hepatic disease. Macrocytosis and the elevated levels of blood fats tended to confirm this. The degree of bilirubinemia and the direct van den Bergh reaction gave no assistance in distinguishing the type of jaundice present. Nevertheless, the presence of the gallstones necessitated exploration and jaundice demanded investigation of the common bile duct. The surgical findings confirmed the preoperative diagnosis.

Increased levels of cholesterol and cholesterol esters in the blood are encountered commonly in obstructive jaundice, but seldom to the degree seen in this case. On the other hand, they are often reduced, at times markedly, in acute hepatic disease. The increase noted in this, an instance of chronic hepatic disease without obstruction of the common bile duct, is occasionally encountered in cases of similar type or in obstructive biliary cirrhosis. The significance of this disturbance is unknown, but apparently has only a slight effect on the surgical risk.

Case VI. The patient, a man, aged sixty-five years, complained of not feeling well for six months prior to registration. For five weeks his appetite had been impaired. Occasionally, he had noted slight discomfort and nausea after eating. Jaundice of painless onset had been noted for two weeks. He had lost thirty pounds (13.6 kg.). Pruritus was not a complaint.

Examination revealed deep jaundice and slight enlargement of the liver. A mass was not palpable in the abdomen. Anemia was not present. Duodenal drainage revealed moderate flow of bile. The value for serum bilirubin measured 18.2 mg. per 100 cc.; the van den Bergh reaction was direct. Excretion of hippuric acid was reduced to 1.4 Gm. Results of the galactose tolerance test were normal. The value of blood cholesterol was 214 mg. and that of cholesterol esters 88 mg., each per 100 cc. of plasma. Prothrombin clotting time of the blood was twenty-four seconds.

After preoperative preparation exploration was performed. The liver was found to be somewhat irregular in shape but was otherwise normal. The gallbladder was collapsed and its wall somewhat thickened; it was opened and drained. The common bile duct was small, but probes passed into the duodenum without meeting any obstruction. In the region of the common hepatic duct, there was possibly some narrowing, which was suspected as being neoplastic. A T-tube was inserted in the common bile duct.

Convalescence was normal. One hundred to 700 cc. of bile drained daily from the common bile duct. The jaundice gradually decreased, but the value of serum bilirubin remained at 4 mg. per 100 cc. during the last two weeks of observation.

Comment. The patient's age and previous good health, the painless onset of deep persisting jaundice and the loss in weight suggested the presence of neoplasm. The presence of bile entering the intestine indicated that obstruction, if any, was incomplete. The degree of jaundice in the absence of complete obstruction, the reduced synthesis of hippuric acid and lowered level of cholesterol esters in the blood pointed to rather severe and acute hepatic involvement. The findings at operation were not conclusive. It was thought that a malignant lesion of the hepatic duct may have been present. However, the drainage tube was inserted in the common bile duct below this level and the external flow of bile was free. The jaundice cleared to a large degree. The common bile duct was not enlarged nor was there obstruction at its lower end. The whole picture can best be explained on the basis of an acute hepatic degenerative process, from which complete recovery may occur or in which improvement may be followed by relapses, progressing to acute atrophy or cirrhosis. Exploration probably served no useful purpose in this case except as an aid in the diagnosis. In a fair number of cases, however, exploration cannot be avoided. Although administration of anesthetic agents and surgical manipulation may aggravate a primary hepatic condition, nevertheless, cases are frequently encountered in which improvement seems to result.

Case VII. A woman, aged sixty-eight years, had had severe, persistent pruritus and mild, painless jaundice for three years. In the ten previous years, there had been occasional attacks of moderate, colicky pain in the right upper quadrant of the abdomen. Slight indigestion had been noted. The gallbladder had been removed elsewhere ten months prior to registration. The patient understood that it had contained muddy material. Drainage had not been established. Convalescence was satisfactory but the symptoms had continued. Weight and strength had been maintained reasonably well.

Physical examination revealed many excoriations, mild jaundice and some enlargement of the liver; the liver itself was smooth. Duodenal drainage revealed an excellent flow of amber-colored bile. Anemia was absent, but generalized macrocytosis was present. The value of serum bilirubin was 5.2 mg. per 100 cc.; the van den Bergh reaction was direct. Prothrombin clotting time of the blood was normal. Excretion of hippuric acid was reduced to 1.4 Gm. The value of cholesterol was 555 mg. and that of cholesterol esters 417 mg., each per 100 cc. of plasma.

On exploration, the liver was enlarged, reddish and somewhat mottled. Moderate hepatitis was present. The common bile duct was normal in size and did not disclose evidence of obstruction. A T-tube was inserted for drainage. One hundred to 185 cc. of bile, which contained large amounts of bile salts, drained daily until the tube was removed on the twenty-first postoperative day. The fistula closed promptly. The jaundice was slightly lessened at the time of dismissal from the clinic. The pruritus was relieved temporarily.

Comment. The onset of this illness associated with severe and persistent pruritus and followed immediately by mild nonfluctuating jaundice indicated a primary hepatic disorder. Further indications were the maintenance of weight and strength, the enlarged liver, apparent absence of biliary obstruction at the time of original operation, as indicated by normal convalescence without development of fistula, and absence of pain or fluctuations in her condition postoperatively. However, the uncertainty of the operative findings and continuation of the jaundice led to further exploratory operation. Tests for hepatic function were inconclusive in distinguishing possible obstruction of the common duct with associated secondary biliary cirrhosis from primary hepatic disease. At operation no pathologic process amenable to surgical relief was encountered. The diagnosis was primary hepatic disease and the prognosis is unfavorable. It probably represented an early stage of cirrhosis in which jaundice and pruritus were prominent symptoms.

SUMMARY

An adequate history and physical examination, determination of the patency of the bile ducts and the use of tests for hepatic function usually will establish the diagnosis as to the type of jaundice present, the nature of the underlying pathologic process, such as stone, stricture, malignant disease, hepatic degeneration or hemolytic process, the possibility of a combination of factors and some knowledge of the functional capacity of the liver. Indications for surgical treatment usually will be manifest, together with some information as to the possibilities of postoperative difficulties.

SPECIFIC INFLAMMATORY DISEASES OF THE LOWER BOWEL

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GENERAL CONSIDERATIONS

TO attempt diagnosis of diseases of the lower bowel by symptomatology is a great mistake. It is not unusual for a patient with cancer of the lower bowel to describe symptoms easily interpreted as being of inflammatory origin; likewise, it is often difficult or impossible to differentiate between organic and functional diseases by symptomatology alone. To attempt diagnosis from symptoms will inevitably lead to error, embarrassment and sometimes tragic results.

These errors can be avoided by examining the patient. The facilities required need not be elaborate or expensive. Simple digital examination, which should be routine in all general examinations, will often disclose valuable information. All patients who have symptoms referable to the lower bowel should have a routine endoscopic examination. For this, the inverted position is preferable because of the advantage gained by partial straightening of the sigmoid due to gravitation and by the tendency of the bowel to balloon which makes observation easier. The lower bowel should be emptied, preferably by repeated plain warm enemas taken two or three hours prior to the examination. A suction apparatus with a long tip is an added advantage in removing excessive mucus or fluid, although in many instances this can be accomplished by using cotton swabs.

Valuable information may often be obtained by accessory examinations. *X*-ray visualization is indispensable in many cases and should be done by one qualified in gastrointestinal fluoroscopy. A flat film of the barium filled colon is sometimes of value but it is essential that this be preceded by fluoroscopic observation as the contrast medium is being introduced into the colon. Thus, filling defects and other abnormalities which sometimes do not show on the film may be detected. It is well to remember that *x*-ray diagnosis in the distal colon (below the pelvic brim) is unreliable, but fortunately this segment of the bowel is easily visualized on endoscopy.

Biopsy specimens for microscopic study may easily be removed during endoscopy. Diagnostic culture and smear specimens are best obtained directly from the lesions during endoscopic examination; if stool specimens are used for this purpose the large amount of extraneous material may unnecessarily complicate and confuse the picture.

The sequence of diagnostic procedures is important. The clinical examination, including endoscopy and removal of biopsy and bacteriologic specimens, should precede the introduction of contrast media into the bowel for *x*-ray study. Endoscopy is likely to increase bowel irritability and spasm. For this reason it is advisable, if possible, to delay *x*-ray examination for twenty-four hours, or longer, following endoscopy.

The anatomic accessibility for examination of the rectum and colon affords opportunity for diagnostic accuracy that exists in few other organs. With reasonable basic knowledge of the physiology and pathology of the intestinal tract, the percentage of accurate diagnoses should be high.

Disorders of the lower bowel can be divided into two general classes: functional and organic. Functional disorders will be disregarded here and organic diseases of inflammatory origin only will be discussed.

Tropical diseases, typhoid fever and certain rare and controversial infections will not be considered.

One or more of the following symptoms should arouse suspicion of organic colonic disease:

1. Increased constipation or looseness of stools.
2. The passage of pathologic fluids, such as blood, pus or excessive mucus.
3. Frank diarrhea, acute or chronic, with or without blood, colic, pain, fever or weight loss.
4. "False alarms," that is, defecatory desire with passage of little but gas, pus, and mucus, with or without blood.
5. Necessity of getting up at night because of persistent desire to defecate.
6. Persistent abdominal pain or tenderness.
7. Loss of weight not otherwise accounted for.

It should always be remembered that colonic or rectal cancer may produce any of these symptoms.

The treatment of these diseases is primarily *medical* although *surgery* is occasionally required, especially when complications arise.

Treatment in general should include careful dietary management and efforts to secure physiological rest. During acute and subacute stages of any colonic infection the patient should remain in bed. Liquid and semisolid foods, without irritating residue, are usually well tolerated during acute stages. Among these are rice, oatmeal and barley gruels, lamb, chicken and beef broths, egg albumen, fermented milk and gelatine. Milk is considered undesirable in most cases. As acute manifestations subside, nonirritating solid foods, such as lean, chopped beef, boiled or poached eggs, toast, custards, chicken and lamb chops may be allowed. Adequate vitamin intake, especially the B group, should not be overlooked. As recovery progresses the diet can be increased. Irritating foods, such as cauliflower, celery, corn, cabbage, fried foods, gravies, raw fruits, lobster, crab, alcohol, and condiments, should not be eaten until recovery is complete.

Symptomatic medication should be used freely. The colon is influenced by psychic factors and every effort should be made to keep the patient physically comfortable and mentally at ease. During acute attacks with distressing symptoms, adequate doses of morphine, or other opiates should be used. Later the barbiturates and other sedatives are useful to promote repose. Demulcents and antispasmodics often serve a useful purpose. Hot stupes are sometimes effective in relieving abdominal distress.

CHRONIC ULCERATIVE COLITIS

This is an inflammatory disease of the colon presenting characteristic pathologic changes, most frequently occurring between the ages of twenty to forty. Its chronic course is often interrupted by acute exacerbations and its effect may be devastating.

The *etiology* is a controversial matter. The excellent work of Bargen and his associates has thrown a great deal of light on the subject. Their belief, based upon convincing laboratory and clinical research, is that it is a specific bacterial disease produced by a diplostreptococcus which they have isolated. Other workers, whose opinions must be respected, have been unable to duplicate Bargen's findings and have advanced other hypotheses of etiology. Thorlakson believes Bacillus dysenteriae to be the cause, while Paulson is inclined to think it is idiopathic; McCarrison, Mackie, and others, believe avitaminosis to be an important factor, and other observers have incriminated various organisms. The bacterial flora of the normal colon is neither simple nor constant and pathologic changes may produce a bewildering complex.

The *pathologic changes* in chronic ulcerative colitis are typical. In probably more than 90 per cent of all cases the lesions first occur in the rectum or lower sigmoid within easy reach of the sigmoidoscope. In the early stages, diffuse inflammation and

edema of the mucosa occur which are followed by the formation of innumerable minute abscesses; these later break and form the tiny ulcers which are so characteristic. As the disease progresses these ulcers may coalesce and, due to secondary infection, form large, ragged, denuded areas. In severe cases, only remnants of mucosa may be recognized.

The inflammatory process extends deep into the bowel wall. It may remain localized to a single segment, or the entire colon may become involved. The mucosa sometimes becomes studded with polypoid growths, perirectal abscesses are not infrequent, and less frequently perforation and fatal peritonitis occur. Visualization by *x*-ray shows the involved bowel to be thickened, narrowed, and shortened, with obliteration of the normal haustral markings.

The early *symptoms* may be mild and later become severe and prostrating, depending on the progress of the disease. Passages of blood, pus and mucus are characteristic, and the number of passages in twenty-four hours may vary from one or two to twenty or thirty or even more. In severe cases most of the movements may consist entirely of bloody mucopus without admixture of feces. Tenesmus, with or without colic, is usually present and may be distressing. As the disease progresses, constitutional symptoms, such as malaise, anorexia, general weakness, low grade fever and loss of weight become evident.

The *diagnosis* is suggested by the symptoms. The proctoscopic picture in most cases is typical as described above in pathologic changes. The ulcerated area is bathed in mucopus and bleeds on slightest trauma. The normally sharp margins of the valves of Houston become thickened and rounded, and lose their normal elasticity. Cultures should be taken with a sterile applicator directly from the ulcerated area and transferred to Rosenow's medium. If the technic described by Bargen is followed meticulously, a diplostreptococcus can be isolated in many cases. This is of diagnostic value and it also can be used in preparing an autogenous vaccine for therapeutic use which is highly desirable. Abdominal tenderness, especially over the sigmoid, is usually present.

The *x-ray findings* indicating thickening, shortening, loss of haustral markings and (in advanced cases) "lead pipe colon," are valuable supplementary evidence and show the extent of the bowel involved.

Treatment must be modified in many instances to suit the individual case. In the acute phases, rest in bed is essential. The diet should be carefully regulated so as to be of ample caloric value, of high vitamin content and free from rough and irritating residue. A careful search for focal infections, and their eradication if found, is important. During the acute stage, or during a severe exacerbation, the use of immune serum is of great value, while vaccine therapy should be used almost routinely in the chronic stage. Autogenous vaccines are preferable but stock vaccines are often used when satisfactory cultures cannot be obtained.

The treatment of chronic ulcerative colitis is essentially medical, but in certain intractable cases, and in the presence of surgical complications, operative interference may be required. Perirectal abscesses demand incision and drainage, while the development of polyposis of any great extent can probably be best managed by electrodesiccation. In extremely severe, advanced and intractable cases, ileostomy may be justified. These patients are poor risks and any surgical procedure involving the intestines should be undertaken with trepidation. In carefully selected rare cases, the formidable operation of colectomy may be considered advisable as a secondary procedure following ileostomy. Appendicostomy, to produce an opening for through-and-through irrigation, has been recommended, but is not in general favor.

Local antiseptic applications, either as irrigations or as retention enemas, are of doubtful value because the inflammatory changes are in the bowel wall and are not influenced by surface applications; further-

more, the trauma incident to administration and distention of the bowel may do more harm than good.

BACILLARY COLITIS

Bacillary colitis, or bacillary dysentery, is an infectious disease of the lower bowel, characterized by acute onset, often with a chill followed by fever, abdominal pain and diarrhea with passage of blood, pus and mucus. While it is more prevalent in the tropics and subtropics, it is not uncommon in the temperate zones.

The *etiologic agent* is Bacillus dysenteriae. Several strains of this organism have been isolated of which those of Shiga and Flexner are most important. In this country children are more apt to be infected by the Flexner strain. The contagion is conveyed directly or indirectly from the excreta of a sufferer of the disease, or from a carrier, to the food or drink of the victim. For this reason the incidence of the disease, like typhoid fever, is in inverse ratio to the effectiveness of general sanitation and personal hygiene.

The *pathology* is essentially that of an ulcerative colitis, with most intensive involvement in the rectum and sigmoid. The early stage is one of acute catarrhal inflammation followed by ulceration. Large areas of mucosa may be destroyed and, with profuse fibrinous exudate, cast off as a pseudomembrane. The acute process is usually limited to the mucosa and submucosa, although in severe cases the bowel wall may be involved. Perforation with peritonitis is a rare complication.

Symptoms appear after an incubation period of three to five days and are usually sudden in onset. There may be an initial chill followed by fever, abdominal pain and diarrhea. Blood may not be seen in the first stools but appears with the onset of ulceration. Tenesmus may be severe if the rectum is the site of extensive ulceration. Abdominal tenderness, especially over the sigmoid area, is usual.

The stools are watery and contain blood and mucopus; they may vary in number from a few to twenty or thirty per day and, when copious, may result in a serious fluid loss. Constitutional symptoms including fever, headache, loss of appetite, malaise, weakness and muscular aches and pains vary in degree, depending upon the severity of the infection. In rare cases of fulminating, virulent, infections, overwhelming toxemia and dehydration may produce delirium, collapse and death.

The *diagnosis* is made by identification of the causative organism early in the disease, and by a positive serologic (agglutination) reaction after the first several days. Cultures to identify Bacillus dysenteriae may be made from mucoid particles taken from a fresh warm stool, or from scrapings taken directly from the ulcer bases through the proctoscope.

Treatment is both general and specific. Isolation and communicable disease precautions, as in typhoid fever, are important. The acute phase of the disease is often somewhat alleviated by the administration of a brisk cathartic such as castor oil or a saline. This clears out the bowel and eliminates much of the toxic material. The diet at first should be nonirritating liquids to which bland solids may gradually be added as conditions justify. Supportive intravenous fluids, with or without transfusions of blood, should be given as the exigency of the case requires. Symptomatic medication should be used freely to make the patient as comfortable as possible. Opiates are highly desirable when the severity of symptoms justify their use. Polyvalent antidysentery serum is considered a specific for this infection. It may be given intramuscularly once or twice daily, or, in urgent cases, it may be given intravenously in smaller doses. The serum should be used in the early stages of the disease. Vaccines, especially autogenous, are preferable for treatment of later chronic manifestations.

AMEBIC COLITIS

Amebic colitis (amebiasis, amebic dysentery) is a specific ulcerative disease of the colon caused by the parasite Endameba

histolytica. It is common in the tropics but may become endemic in temperate climates as was the case in Chicago a few years ago.

The *etiologic* parasite produces the disease while in the motile, or vegetative form, but it is transmitted only while in the cystic stage. Transmission occurs when these cysts, discharged from the intestine, contaminate drink or food consumed by others. Vegetables, such as lettuce, may be contaminated in the field by excreta from an active case, or from a carrier. A food handler, who is a carrier, may disseminate the disease widely. It is probable that 20 per cent of the people who swallow amebic cysts will develop amebiasis, the incubation period of which varies from three weeks to a few months.

Pathologic changes are most pronounced in the proximal colon; occasionally the transverse colon, the descending colon or the rectum are affected. The parasite penetrates the colonic mucosa producing tissue necrosis and ulceration. The ulcers do not show typical inflammatory response; leucocytic infiltration does not occur as with bacterial invasion. The ulcers undermine adjacent mucosa, and they may become confluent, leaving only shreds of mucous membrane attached to the bowel wall. In chronic cases, fibrous tissue replacement in the submucosa and mucularis may cause thickening and contraction even to the point of stricture formation. Granulomatous masses occur infrequently and may easily be confused with cancer.

The parasite may penetrate the bowel wall, gain entrance to radicles of the portal vein and be carried to the liver where solitary or multiple abscesses may be formed. Perforation and serious hemorrhage are rare complications.

Symptoms are variable. While dysentery is the most common symptom in the acute, virulent type of infestation, in milder and chronic cases constipation or vague abdominal distress may be the outstanding complaint. Dysentery is characterized by passage of liquid or mushy stools which may vary in number from two or three daily to almost continuous evacuation, depending upon severity of the case. Pus and blood-streaked mucus are usually in the stools. Abdominal cramps and soreness are often present. When the acute stage subsides, a stage of equilibrium between the parasite and host may be reached only to be upset at intervals by catharsis, dietary or alcoholic indiscretions, or general causes. Liver abscess will produce pain in right upper abdominal quadrant, radiating to the right shoulder. Chills and fever, often resembling malaria, may be present. An unproductive cough may be the result of diaphragmatic irritation.

Diagnosis in the dysentery stage is made by finding the motile specific parasite in fresh stools or in scrapings from ulcers; in the chronic phase demonstration of amebic cysts is required. Liver abscess should be suspected when an amebiasis patient develops right upper abdominal pain and enlargement of the liver. The initial liver-abscess drainage does not show the parasites but often they can be found in scrapings from the abscess walls. Liver abscess occurs in 15 to 20 per cent of active amebiasis cases.

The *prognosis* is favorable with early recognition and prompt treatment. The development of complications greatly reduces the chance of recovery.

Treatment. Ipecac is a time honored drug in the treatment of amebiasis. Its alkaloid, emetine, is preferable and is usually employed in doses of 1 gr. daily administered subcutaneously. It is especially effective in relieving the distressing symptoms of the acute stage of the disease. It should not be used longer than a period of eight to ten days because of the possibility of producing myocardial damage.

Certain arsenicals are considered specific in the treatment of amebiasis; of these, carbarsone is generally considered the most effective. Its dosage is 4 gr. three times daily by mouth, with a rest period after ten days of treatment. Iodoxyquinolin sulfonic acid (yatren, chiniofon, anayodin), an

iodine compound, is also an effective chemotherapeutic agent. It is given by mouth in doses of 7½ to 15 gr. three times daily for seven to fourteen days. Some clinicians prefer a treatment régime essentially as follows: (1) emetine subcutaneously for seven to ten days to control acute symptoms, followed by (2) carbarsone by mouth for seven to ten days, followed by (3) yatren by mouth for seven to ten days. Then the series of carbarsone and yatren may be continued alternately until the patient is found to be free from the disease as demonstrated by repeated stool examinations.

During the acute stage the patient should remain in bed, and the diet, liquid at first, should be discreetly increased as tolerated, using due care to avoid rough or irritating residue. Symptomatic medication, including opiates, should be used as indicated.

Surgical treatment is limited to urgent complications and to the drainage of abscesses. Patients with amebiasis tolerate surgery poorly and procedures which involve handling the intestines carry a high mortality rate.

GRANULOMATOUS INFLAMMATORY DISEASES OF THE COLON

Tuberculosis is the most frequent of the granulomatous infections involving the lower bowel. It is a specific infection caused by the tubercle bacillus, and is most frequently seen in patients who have pulmonary tuberculosis with cavitation, the infectious agent being conveyed in swallowed sputum. Adults seem to be about three times more susceptible than children to this secondary manifestation of the disease. Two forms of tuberculous enterocolitis are recognized: namely, (1) hyperplastic, and (2) ulcerative.

Hyperplastic tuberculosis, or tuberculoma, is most frequently seen in the ileocolic segment of the bowel. It is essentially fibrous hyperplasia rather than caseation as is seen in the ulcerative form. Fibrosis and contraction may cause obstruction of varying degree. It is more frequent in the age group of twenty to thirty years, and is more frequent in Europe than in the United States.

The *ulcerative* form is caused by small, submucous tubercles which undergo caseation and rupture into the lumen of the bowel-producing craters with ragged, undermined margins surrounded by inflammatory areas. Healing and destruction may progress simultaneously. The destructive process usually follows the lymphatics, producing elongated ulcers transverse to the long axis of the bowel. If the rate of destruction exceeds that of healing, large confluent ulcers will occur, and possibly perforation. Perforation may produce peritonitis, or, if omentum or other abdominal structures seal the opening, inflammatory masses may result. Almost all cases of tuberculous ulcerative enterocolitis are secondary to foci elsewhere, usually in the lungs.

Symptoms are usually mild or may be absent in the hyperplastic form until some degree of obstruction is produced. Postprandial abdominal pain, often colicky but sometimes dull and aching, us usually the first and most constant symptom. It may be generalized throughout the abdomen or localized in the lower right quadrant. The clinical picture is not infrequently confused with that of appendicitis. Loss of weight is another frequent occurrence and a palpable tumor is frequently present in advanced cases. Less frequent symptoms are vague digestive disturbances and periods of diarrhea or loose stools which may alternate with constipation. Proctoscopy is usually of little value in the diagnosis of tuberculoma because the lesion is rarely within view. *X*-ray study, after a barium enema, is the most useful diagnostic procedure. The condition may be confused with cancer, which it grossly resembles; but a tuberculoma is usually seen in a tuberculous patient in whom the blood picture seldom shows much change, while cancer of the right colon almost invariably produces a definite, if not profound, anemia.

Ulcerative, tuberculous enterocolitis should be suspected when a tuberculous patient begins to complain of nervous irritability, anorexia or any digestive disturbance. These symptoms usually precede the later symptoms of diarrhea and abdominal pain. Constipation may be present or it may alternate with attacks of diarrhea. Gross blood in the stools is not common, but occult blood can nearly always be detected. The proctoscopic picture, if the ulcers are within view, is often suggestive but may not be conclusive. The ulceration of amebiasis can be excluded by stool examination or by microscopy of scrapings from bases of the ulcers. Biopsy specimens often give valuable information. All clinical evidence, including *x*-ray, in a given case should leave little doubt as to the nature of the disease.

The *treatment* of intestinal tuberculosis is essentially the treatment of tuberculosis elsewhere. Rest and a high caloric nonirritating diet, with adequate vitamin intake, are fundamental. Intestinal tuberculoma, unless mild, is a surgical disease, and the operation of choice is usually a short circuiting procedure, followed by resection of the diseased portion of bowel, preferably in one stage. Surgical intervention is rarely justified in the absence of obstructive manifestations. Ulcerative tuberculous enterocolitis becomes surgical only when complications, such as perforation, abscess or stricture develop.

Actinomycosis is a specific, destructive, inflammatory disease, caused by Actinomyces hominis (ray fungus) which sometimes invades the lower intestinal tract, the most frequent site being the ileocolic region. It is prone to spread by direct continuity of tissue rather than by the lymphatics or blood stream. Abscesses, which may invade the abdominal wall, followed by draining sinuses or fistulae, are not infrequent complications. In advanced cases the prognosis is unfavorable and the mortality rate is high; in early cases the outlook is more favorable. Large doses of potassium iodide, and perhaps radiotherapy, seem to offer the best hope of cure. When anatomic localization permits, surgical extirpation probably offers the best chance of cure.

Syphilis of the colon and rectum, because of its rarity, is of comparatively little clinical importance. Early lesions, including mucous patches, are occasionally seen about the anus. Gummatous lesions involving the rectum and colon are extremely rare. Benign stricture of the rectum was formerly thought to be of syphilitic origin but it is now known to be a distinct clinical entity.

Lymphogranuloma venereum and rectal stricture is a specific inflammatory disease caused by a filtrable virus, which is usually transmitted by sexual contact. While it is primarily a genital infection, it is now known to be the causative factor in the vast majority of cases of benign stricture of the rectum.

Lymphogranuloma venereum (lymphopathia venereum, lymphogranuloma inguinale, climatic bubo, tropical bubo, strumous bubo, Durand-Nicolas-Favre disease, the fourth venereal disease, the sixth venereal disease, nonvenereal bubo, etc.) and its rectal manifestations are seen most frequently in the lower strata of society in which the incidence of venereal diseases is highest. Negroes seem to be peculiarly susceptible and the age limits correspond generally with the period of greatest sexual activity. Negro women constitute about 80 per cent of all cases.

The primary genital lesion may be so inconspicuous as to go unnoticed. The contagion has a predilection for lymphatics and it spreads along these channels. In the male, the inguinal nodes become involved, producing characteristic buboes; in the female the perirectal lymphatics are invaded and the characteristic rectal manifestations of the disease are produced.

Three *stages* of rectal lymphogranuloma venereum are recognized: (1) Proctitis with diffuse inflammation of the bowel wall usually accompanied by ulceration of the mucosa and the passage of blood, pus and

mucus. Perirectal abscesses and fistulae are not unusual complications. (2) Proctitis obliterans (prestenotic) follows the early proctitis and is characterized by diffuse thickening of the rectal wall which resembles a thick, wet, rough leather tube. Ulceration is more extensive and there is an abundant discharge of a mixture of blood and pus which, in appearance, is not unlike cream of tomato soup. (3) Fibrous stricture of the rectum follows, and is the result of the extensive, inflammatory invasion of the rectal wall. These strictures are usually tubular and extend from just above the anus almost to the peritoneal reflection. Rarely will the process extend upward and involve the sigmoid and descending colon. These advanced fibrous strictures, when palpated through the posterior vaginal wall, feel not unlike a segment of broom handle. The lumen becomes smaller as the stricture progresses and it is not unusual for the rectal canal to be no larger than a lead pencil. At this stage, ulceration is extensive, only fragments of mucosa remaining, and purulent drainage is profuse.

Chronic partial obstruction increases as the stricture develops. Secondary infection, with the obstruction, produces symptoms of toxic absorption.

Symptoms become increasingly severe as the pathologic changes progress. In the early stages, frequent passages of mucus, blood and pus occur, accompanied by more or less tenesmus. Perineal swelling and pain are complained of when complicating abscesses develop. As partial obstruction becomes more evident, systemic symptoms such as malaise, meteorism, anorexia, low grade fever, loss of weight and general impairment of health appear. Often there is an almost constant dribbling from the anus of an irritating, excoriating discharge which adds to the patient's misery.

The *diagnosis* is almost self evident if the disease is kept in mind. The possibility of cancer usually can be excluded by clinical characteristics but occasionally biopsy study is required. The Frei test is specific for lymphogranuloma venereum. This test is based upon the allergic skin response to a small amount (0.1 cc.) of a heat inactivated saline suspension of pus from a lymphogranulomatous bubo (or antigen from other sources) when injected intradermally.

Treatment of the disease is not always satisfactory. The use of Frei antigen intradermally once or twice weekly will sometimes delay or even stop its progress in the early stages, but stricture formation is an irreversible process. Mild antiseptic irrigations increase the patient's comfort but have no curative effect. Cutting and dilating the stricture have been disappointing because the stenosis recurs. Application of carbon dioxide snow and the use of heat in the form of diathermy have been beneficial in some cases. When obstructive symptoms occur, colostomy gives immediate and often permanent relief. Complicating abscesses should be incised and drained, and in some cases, fistulectomy is desirable.

RADIATION PROCTITIS

This is a lesion, sometimes called facticial proctitis, caused by the destructive action of radioactive energy, most frequently seen in women who have had radium therapy for cancer of the cervix uteri. It begins as an edematous thickening of the rectal mucosa, almost always on the anterior wall opposite the site of radium application. If the amount of radiation absorbed has been sufficiently small, this reaction may subside gradually without actual tissue destruction. As a rule, however, the stage of edema is followed by necrosis and ulceration. The mucosa and submucosa slough away, leaving a pearly white fibrous base sharply circumscribed by reddened, hyperemic mucosa in the margin of which the blood vessels stand out prominently. This hyperemic area bleeds easily on slight trauma. The ulcer may be small and involve only the anterior wall or it may extend entirely around the circumference of the bowel.

Symptoms usually appear three or four weeks after radiation therapy although several months may elapse before the condition becomes apparent. The symptoms are those of ulceration and bowel irritability. The first complaints are usually discomfort or pain and the passage of blood and mucus. Later, tenesmus may become distressing. With a history of radiation therapy and the characteristic appearance of the ulcer there should be little difficulty in arriving at the diagnosis.

Treatment in uncomplicated cases is palliative. Rest in bed and warm, cleansing irrigations are helpful. The tenesmus, if severe, may require an opiate, but soothing instillations of warm olive oil or of starch gruel often give relief. The patient should be instructed to take a warm, plain water enema for its cleansing effect after each evacuation. The direct application of 1 or 2 per cent gentian violet solution or of balsam of Peru through the proctoscope every second or third day may be beneficial. If pain becomes intractable or if stricture develops, colostomy may be required. Posterior proctotomy for stricture is usually unsatisfactory. It relieves the stenosis only temporarily and subsequent contraction is almost certain. If the stricture is severe enough to warrant surgery, it is probably better to do a colostomy.

"Itching" sensation of the skin may be very distressing. It may be associated with an obvious dermatitis or other skin lesion, and then cause and effect are definite. There are, however, many cases where there is no visible cause for the sensation, and then it is termed "pruritus."

PROCTOSCOPIC RECOGNITION OF RECTOSIGMOIDAL AND SIGMOIDAL LESIONS

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THE recognition of rectosigmoidal and sigmoidal lesions is, or should be dependent upon direct or indirect visualization. Anamnesis as related to diseases of these areas is helpful but cannot be depended upon to too great a degree. When the patient's statement that he has experienced bleeding from the anorectal region and that he has hemorrhoids, which is the most common anorectal complaint, is taken too literally, it often results tragically for the subject and proves to be embarrassing to the attending physician. Too often the physician agrees with the patient's diagnosis and without benefit of examination palliative measures are advised. Bleeding is common to many lesions in this area. It is the most frequent symptom complained of by people who have hemorrhoids and also those who have cancer of the lower bowel. If adequate search consisting of thorough sigmoidoscopic examination, and roentgenologic examination if indicated, are not done, then valuable time may be lost in attacking diseases of this area. The symptoms of cancer in this region are the symptoms of complication. Frequently, before subjective symptoms become severe enough to cause the patient with cancer of the colon to seek advice, there is a visually recognizable lesion present. This argues for more frequent use of the sigmoidoscope, which in proper hands can reach past the area where the greater number of malignant lesions of the large bowel are located.

Direct visualization obtained through the skillful use of the sigmoidoscope is of course the most certain means of diagnosis of lesions within its reach. The next best, and an adjunct of increasing help and specificity, is indirect visualization by means of roentgenoscopy and roentgenography. Bacteriology is of course an important aid in ulcerative lesions. Sigmoidoscopy should be employed, however, in all cases for frequently two different entities may be present at the same time. Great strides have been made in roentgen diagnosis of diseases of the large bowel since the advent of the contrast enema and the lateral position for visualizing the lowermost portions of the colon and rectum. The mutual aid and the overlapping of the fields of the proctoscopist and the roentgenologist are not to be underestimated.

Through some chain of events it has so come about that probably the most serious and most frequent organic changes occurring in the colon are within visual reach of one trained in the proper use of the sigmoidoscope. These changes can either be diagnosed at the time of visualization or important aid can be given to assist in their final diagnosis. It is doubtful that much information can be gained by the occasional peeper through the short, unlighted anoscope or rectoscope, which is the usual limit in armanentarium of proctoscopic instruments. If one is to accept the responsibility for diagnosing and treating diseases of this area, it is necessary to have as complete a knowledge of this field as is required of the cystoscopist and the bronchoscopist in their fields. It is true that occasionally the use of the usual poorly lighted rectoscope which is not equipped for inflation, in an unprepared bowel, may reveal a carcinoma or even a small polyp in the rectum. Much, however, is left to chance. It may easily fail to show an early lesion and this is the stage at which diagnosis is most important if the

patient is to have the best chance of recovery.

It is first of all important that one be totally familiar with all variations of size, color, shape, etc., of the so-called normal bowel; for unless one is entirely familiar with the normal bowel and its non-pathologic variations, it will be difficult to know the abnormal in its early form.

Obviously, it is also important that one be equipped properly for the examination of the areas under discussion. Few, if any, physicians doing a general practice have added bronchoscopes or cystoscopes to their equipment, yet there are perhaps still too many who are satisfied if the passage of a rectoscope into an unprepared bowel fails to reveal anything. Being equipped properly entails the possession of an instrument of correct length and it is important that this instrument be sufficiently lighted. It is just as easy to visualize the rectum through a sigmoidoscope as through a rectoscope and the sigmoidoscope has the advantage of enough length to reach the sigmoid where such a large number of changes take place. The question of where the light is placed in relation to the ends of the instrument is one of choice provided that direct and not indirect light is used. It has been the writer's observation, that on numerous occasions decisions made with reflected light, or a proximally lighted instrument have been proved erroneous when a distally lighted instrument was used. It would be reasonable to assume that the closer an adequate light is to the object, the better the definition. Along with the proper instrument, an inflating bulb should be used freely. Frequently in all positions, but to a lesser degree in the inverted proctoscopic position, the bowel is found to be collapsed when the instrument is inserted and thus it is difficult to see between the folds and for this reason, small lesions and early changes in the mucosal pattern may be missed. With the inflating bulb, the bowel wall can be smoothed out and readily studied.

The position of the patient for examination is also one of choice; however, it appears evident that the inverted position, best obtained on a proctoscopic table, has many desirable features. This position allows the abdominal contents to fall away from the pelvis and the lower bowel is, by reason of this, straightened out as much as its anatomical attachments will allow. The introduction of the instrument allows air to pass into the bowel and in this position this alone frequently causes the bowel to balloon out. This factor and the fact that the remainder of the abdominal contents are not pressing on the wall of the lower bowel, tend to smooth out the folds so that inspection of the bowel wall is more adequate. Even in this position, the inflating bulb is often necessary for a clear cut view of all of the surface area and its judicious use is of great aid. Certainly it is more desirable to visualize an area directly then indirectly. Thus, although fluoroscopic study and roentgenograms are of great aid in the diagnosis of disease of the lower colon, it is self evident that a direct view, by one properly trained in the use of the sigmoidoscope, is of far greater value than an indirect view by means of roentgen rays.

It is doubtful if any great value can be placed on digital examination, although this should always precede instrumentation. The knowledge obtained by digital examination, as to whether or not there may be a growth or an obstruction within reach of the finger, the presence of which would necessitate extra care in the introduction of the instrument, is useful. The tone of the sphincter muscles, and if a tumor is present, whether or not fixation has taken place can also be determined. The degree of fixation, however, can be determined about as readily by pressure of the instrument against the growth. It is true that to one thoroughly trained in anorectal diagnosis "the feel" of a carcinoma of the lower bowel is almost diagnostic; however, "the sight" of one is definitely more certain.

The sigmoidoscopic recognition of lesions of the rectosigmoid and sigmoid is dependent, therefore, on a clear unobstructed view of these regions. To obtain this, the lower bowel should be thoroughly cleansed. This is best obtained by means of nonirritating cleansing enemas given a few hours before examination. It is of no value and often a disadvantage to purge the patient. If irritating enemas are used, such as those which contain soap, the mucosa may be inflamed and a false impression gained.

With the patient in the chosen position, a digital examination is done for reasons stated above. The sigmoidoscope with obturator in place is introduced only through the external sphincter mechanism. When this point is reached, the obturator should be withdrawn and further passage accomplished under direct vision. At this time fecal material or retained material from the enema may be encountered. Here it is of inestimable value to have an adequate suction to remove the material through the sigmoidoscope. The passage of the scope under direct vision gently carried to its fulfilment, should never cause a bowel to be perforated. Even though it may be a point of pride with many proctologists to be able to follow the circuitous route from the rectum to the sigmoid without the aid of an inflating bulb to balloon out the bowel, the one using the inflating bulb will be able better to visualize small lesions which might be hidden by folds of the mucosa and the time consumed in the examination may be materially lessened.

It is good practice to concentrate at first on inserting the instrument its full length as quickly as possible and do a detail study as the instrument is being withdrawn. If, with an apprehensive nervous patient, too much time is taken up as the instrument is being inserted, the patient may begin to strain and it may become impossible to pass the instrument its full length.

The recognition of diseases, such as hemorrhoids, fissures, fistulas, etc., which occur in and around the anorectal junction do not come within the scope of this paper. Such diseases as chronic ulcerative colitis in its several forms, one-third of the cases of amebic infection which have lesions that are directly visible in the lower bowel, lymphopathia venereum, tuberculosis, malignant lesions, etc., all are seen most frequently in the rectum but may occur in or extend higher in the large bowel. Such clear cut descriptions of all of these diseases have appeared in the medical literature so often that the writer considers it unnecessary repetition to detail their symptoms and their visual and roentgenological characteristics.

It is the intention of the writer to encourage the more frequent use of the chief diagnostic aids in the diagnosis of lesions of the colon and rectum and to make a plea for the more careful differentiation of organic and functional disease of the colon. He wishes to reiterate only the findings in some of the less commonly described lesions which are being found in increasing numbers in this area. That these lesions are being found more often is due, it is thought, not to so much an actual increase of these lesions but to the fact that the examination of this area is done more frequently and more skilfully.

It has not been the writer's observation that inflammatory lesions of the lower bowel are very common. Vast numbers of patients will say, or say that they have been told, that they have colitis. Careful questioning will bring out the fact that what is usually meant is some alteration of bowel function usually brought on by "doctoring the bowels." This results in vague complaints of gas, abdominal soreness, and the vicious circle of taking something to force elimination, with the result that they become chronically unwell and are good subjects for the "colon irrigators" and such. Careful examination of most of these poor unfortunates fails to reveal anything of an inflammatory or an organic nature.

The most frequent lesions the writer has encountered in his practice which is solely proctology, other than lesions which

occur in and around the anorectal ring, are polypoid lesions of the rectum, rectosigmoid and sigmoid. These have been seen in approximately 5 per cent of the cases observed, all of which have been examined under as ideal conditions as can be obtained. The size has ranged from a few millimeters to several centimeters. Most of these lesions are asymptomatic and are incidental findings resulting from a thorough sigmoidoscopic examination on all patients, regardless of their chief complaint.

There is no typical history given by patients who are found to harbor a polypoid lesion in this region. The majority of the writer's series of polypoid lesions have been incidental findings as stated above. These patients present themselves with complaints ranging from pruritus ani to violent tenesmus. The symptoms presented by such lesions are usually in direct proportion to the size and location of the tumor.

Small sessile lesions can hardly be suspected of presenting symptoms other than bleeding. Bleeding from such a lesion may accompany bleeding from the region of the anorectal ring. Unless careful search is made of the area through which a sigmoidoscope may be passed and if indicated a contrast enema done, it is evident that these lesions may be overlooked and the whole blame be laid on the anorectal region, until the polyp has reached sufficient size to give other symptoms of itself. That this has not infrequently been done is evidenced by the fact that such a large number of cases with even a full-blown carcinoma of the lower bowel give a history of having received treatment for anorectal lesions. After recovery from the anorectal treatment they still have more or less the same complaints and a more thorough examination reveals an additional lesion or lesions. Had these cases been thoroughly examined and given the benefit of a sigmoidoscopic and/or a roentgenological examination, it might have been possible to avert this situation.

The pedunculated lesions by reason of their being pedunculated more often give rise to symptoms. This may be due to several factors. They get in the fecal stream, are irritated and traumatized more readily than is the sessile group, and thus the symptoms of bleeding may occur earlier. Tenesmus resulting from the peristalic waves trying to expel the mass with resulting traction on the mass, may cause pain as well as bleeding. A patient whose history includes such complaints as tenesmus, the occasional passage of blood-stained mucus, vague pains in the abdomen or back, a frequent urge to defecate usually with little results, and a feeling at times as if the rectum were full, with no result from attempts to empty it, should be thoroughly investigated, sigmoidoscopically and with the contrast barium enema. These symptoms may be the warning of a mass fixed low enough so that it can be visualized directly; it may be either a pedunculated or a sessile mass which at times prolapses down into view; or it may be a mass attached too high to be visualized directly and should be searched for by means of a contrast enema. One should guard against the too frequent willingness to be satisfied that what anorectal lesions are seen on casual examination, account for the complaints of the patient.

Polypoid lesions, although innocent appearing at times, frequently show microscopic changes ranging from hyperplasia or anaplasia to frank malignancy. Of the writer's series approximately 70 per cent showed such variations from the normal. It appears illogical to say that only a certain percentage of these growths become malignant when so much incriminating evidence has been piled up against them. An analysis of the writer's series of polypoid lesions leads one to assume that if an individual has a polypoid lesion of the colon or rectum, and lives ten years, his chance of having a carcinoma in the corresponding location is too great to be minimized.

The polypoid lesions which are found to be benign are most frequently adenomas; however, lipomas and fibromas occur infrequently as intraluminal tumors. As a rule types other than the adenomas occur higher in the colon, are pedunculated and for this reason are more prone to prolapse causing more or less a degree of intussusception with resulting symptoms. At the time of sigmoidoscopic examination, these masses may have returned to their original position and will not be seen. Their diagnosis then, becomes dependent on visualization by roentgen examination. This is best accomplished by a fluoroscopic observation of a barium enema followed by a contrast enema.

Not infrequently patients present themselves with complaints of fulness in the rectum, of infrequent defecation, others of frequent emissions of small amounts of bowel content, burning, backache and other vague rectal symptoms and are found to have a mass in or above the rectouterine or rectovesical space. This can not always be felt by digital examination; however, the passage of the sigmoidoscope into the rectosigmoid, or higher, is impeded by a mass which impinges on the lumen thus fixing the upper rectum and rectosigmoid so firmly that the passage of an instrument above this is impossible. The finding of such a mass is added evidence of the importance of routine sigmoidoscopic examination because it frequently indicates primary disease in other parts. The mass, may be due to implants from malignant disease of the stomach, small bowel, colon or genitourinary tract. Not all of these lesions are secondary growths from malignancy in other locations. They may be the result of inflammatory or benign lesions of the gastrointestinal, urinary or genital organs, impinging on the rectal wall. The finding of such a lesion means that careful investigation should be made of the other systems. The secondary malignant lesions are usually localized anteriorly in the cul-de-sac and are usually in one mass. The secondary inflammatory manifestations may be made up of several masses, giving the lumen a nodular appearance and are not as universally located anteriorly as are the malignant implants.

One of the writer's four cases which presented a great number of nodular masses protruding into the lumen of the rectum and rectosigmoid, over which the mucosa was intact, was found to have a tuberculous peritonitis on exploration. The biopsy from one of the nodules taken through the rectal wall, showed only inflammatory tissue. The remaining three patients did not submit to operation, but it is believed that probably all were tuberculous.

Some patients who seek medical advice on account of changes in bowel habit, usually a frequency of stool, are found to have a condition in their lower bowel which when viewed through a sigmoidoscope, is difficult to describe. Usually these patients complain of anorexia, gas, an increased number of stools which are usually soft and mushy if not watery, vague abdominal pain and soreness, muscular pains in the legs and nervousness. These individuals are frequently on a self imposed, inadequate diet, have a slick tongue, are nervous and very introspective and frequently have generalized tenderness to palpation of the abdomen. The lower colon and rectum, when viewed through a sigmoidoscope, are found to be rather atonic and relaxed. The mucosa is moist, reddened, although definitely not ulcerated, and very smooth. The roentgenogram of the colon reveals nothing as a rule, but on following the barium meal through the small bowel, changes are frequently seen in the mucosal pattern which leads one to believe that a dietary deficiency is present. It is perhaps true that some of the ulcerative lesions, which one sees localized in the lower colon but may be found in other parts of the colon by roentgenograms, in which no bacterial cause can be found, are advanced stages of this same process. These lesions are limited to definite segments of the bowel

and do not present the same picture as thrombo-obliterative colitis which has been so frequently described and which is undoubtedly bacterial in origin. Why this lesion, if it be the result of deficiency, should be localized to segments of the colon is not now known.

The ability to distinguish between organic and functional disease of the large bowel is the most necessary part of the armamentatium of those who examine this portion of the body. The diagnosis colitis is still probably one of the most abused in medical literature. The term colitis as it is commonly applied, is closely akin to the miasmatic group of diseases and too frequently it is the waste basket into which is thrown many of the vague complaints referable to the abdomen with or without change in bowel habit. There is a vast army of unfortunates being treated solely on this basis. Many have never had the benefit of a thorough colonic examination. They are frequently on inadequate diets, either prescribed or self imposed. They are already "doctoring their bowels" or are advised to do so either with laxatives or lavages or both, until the normal physiology of the bowel and their nervous system are so upset that they are burdens to themselves and to their families. Exhaustive studies of those individuals are essential. In the face of negative results, it requires tact and patience to convince these sufferers that their symptoms, as related to the colon, are not the result of organic disease but the result of a disordered physiology due either to disease in other parts, or are the result of treating symptoms which are not related to the colon or are functional in character. To stop this type from their use of laxatives, mineral oils, enemas, and fadistic diets, is often a long and arduous task.

The skilful use of the sigmoidoscope should become more general, for often only by its use can the presence of early organic disease be determined or ruled out. Early diagnosis of colonic lesions is important and can be easily made by skilful use of the diagnostic aids at our command.

ANAL FISTULA AND ABSCESS

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THE term, anal fistula or abscess, is not an accurate anatomical description of the lesions usually classified under these titles. In most instances fistulas or abscesses of this type have their origin and primary opening in one of the crypts of Morgagni, which are located in the terminal-most part of the rectum, and therefore the lesion is not strictly anal in character. The external or secondary openings may be anywhere in the skin adjacent to the anus. Regardless of terminology this is the lesion present in approximately 95 per cent of all patients presenting fistulous openings about the anus. While this is usually the case, it is important to remember that occasionally an abscess or fistulous opening in this region may have its origin in some distant point, as the female organs, the prostate, seminal vesicles, posterior urethra, in an infected sigmoid diverticulum, Pott's disease, pelvic infections, perirectal infections, perirectal cysts, appendiceal abscess, and other less frequent conditions.

As has been stated, the origin of a fistula or abscess, commonly termed an anal fistula or anal abscess, is usually from an infection involving a crypt of Morgagni. Tucker and others have pointed out the presence of preformed ducts connecting with the base of these crypts and extending outward into the perianal tissues. He believes the infection develops in these preformed ducts rather than in the crypts themselves. I have not been able to convince myself that this is true, at least not in all cases. Tucker has shown microscopically that these ducts do exist in certain individuals, but I am inclined to believe that further study will show that these ducts are a vestigial remnant, that their presence or absence is as variable as is the case of pilonidal sinus, and that they are not present in all persons. At least I believe that an infected crypt of Morgagni is most often the cause of perianal abscess formation. There is certainly a great variation in the crypts of Morgagni. In some cases these crypts are represented by the merest depression in the rectal mucosa with no pouch or pocket at their terminal ends. In others there is a definite pouch or pocket at their caudal termination. This pocket may be a centimeter or more in depth, extending beneath the epithelium of the anal canal caudal to the pectinate line. These pouches being the terminal portion of the crypts of Morgagni, are lined with rectal rather than anal epithelium. There is no doubt that an anal fistula occurs much more frequently in persons having well developed crypts with deep pockets at the caudal end, than in those in whom the crypts are rudimentary and the pouches are very small or absent.

The usual sequence of events in the formation of an anal fistula is infection of the mucosa lining the crypt, extension of the infection through the bowel wall, the formation of a perianal abscess and then rupture or incision of the abscess, which completes the fistula. The only instances in which infection of the crypt does not precede abscess formation, is when there is perforation of the crypt wall by a foreign body. This is a fairly common occurrence due to the ingestion of sharp spicules of bone, toothpicks, etc. While more than one crypt is often infected, usually only one is involved in an infective process severe enough to extend through the bowel wall. For this reason, at operation, a fistula-in-ano is seldom found to have more than one internal opening. Occasionally, a perianal abscess may occur without actual perforation of the bowel wall. In this case, exten-

sion is directly through the rectal wall or by way of the lymphatics. When abscess occurs and a fistula does not result, it is either due to infection spreading in this manner or there has been a very small perforation, which healed after the abscess was drained externally. In the vast majority of instances there is a well defined opening through the bowel wall and a fistula results. The immediate causes of the extension of the infection through the viscus varies. In some cases the infection may simply gradually progress until perforation occurs. In many instances, I believe, the cause is some unusual traumatization, occurring in a crypt already weakened and made friable by previously existing infection. This trauma may be caused by the passage of a large hard stool, the excessive muscular action of repeated defecation due to an acute diarrhea, or some external or instrumental injury. The rapidity of formation of the abscess, once the infection has extended to the perianal tissue, depends upon the type and virulence of the organism and perhaps the resistance of the host. These abscesses usually show mixed infection, although occasionally an almost pure culture of Bacillus coli, a short chain streptococcus, the tubercle bacillus or other organisms may be found. In most instances, once perforation of the bowel wall occurs, the abscess develops within a few days, with the usual redness, heat and pain. There is, in varying degrees, a rise in temperature, a leukocytosis, a feeling of malaise and often some muscular aching. Pelvic and inguinal lymph-nodes may be involved. In rare instances the infection may be of such virulence that it quickly spreads throughout the body and death results. I recall one case in which death occurred within three days and there was involvement of many lymph-nodes, multiple abscesses of the lungs and liver, and a generalized blood stream infection. The other extreme is the presence of a practically nontender area of induration adjacent to the anus which is the first symptom noted by the patient. This hardened mass gradually approaches the skin surface, slowly increasing in size, finally becomes slightly reddened and tender and eventually ruptures or is incised. If spontaneous rupture is allowed to occur, the entire cycle may take several weeks. This is the description of an abscess caused by an organism of extremely low virulence. This description also coincides with that caused by the development of an abscess due to the tubercle bacillus. The difference is, that with a pyogenic organism, rupture or incision of the abscess discloses the presence of ordinary pus, while in a tuberculous condition, unless contamination by other organisms has occurred, discharge is thin, watery and flocculent.

Occasionally, abscesses originating in a crypt of Morgagni do not rupture perianally. In this event, one of two types of syndromes may develop. In the first, an indurated area is felt adjacent to the anus. This is more or less sensitive and may or may not, present evidence of superficial inflammation. After a time these symptoms disappear spontaneously, and usually rather suddenly. This is the result of the abscess having drained into the rectum through the primary opening. In the other variety the patient notes a deep-seated soreness in the anal region but without external evidence of induration or inflammation. Soreness is increased by pressure or contracture of the muscles in this region. These symptoms also disappear spontaneously when the abscess drains internally. In either case the cycle may repeat itself many times without the abscess rupturing externally. In some instances, external rupture eventually may occur, or the abscess present itself so superficially that it is incised. This type of abscess and sinus not infrequently acts as a definite focal infection, serving as a spark plug for a more distant infectious process.

While most abscesses and their resultant sinuses and fistulas result from infections in the crypts of Morgagni, such conditions can and do result from infections in the

wall of the anal canal. The most common infectious processes are chronic fissure-in-ano, or recurrent anal cracks and abrasions. A congenitally small anal canal or an acquired anal stenosis predisposes to these conditions. The abscess resulting from infection in this region is usually more superficial as is the resulting fistulas. For this reason operation upon fistulas of this origin does not necessitate the division of the external sphincter muscle as often as in the case of fistulas originating in a crypt of Morgagni.

When it becomes necessary to operate upon an abscess or fistula, it is a matter of importance whether the infectious process has reached the ischiorectal fossa by extension *through* the space between the internal and external sphincteric muscle, or has reached it by extension *around* the external sphincter. In the former case, the external sphincter must be divided in order to cure the fistula; in the latter, division is not necessary. The division of the external sphincter is not as vital a matter as is sometimes supposed, since it actually has little to do with what is loosely called "control of the bowels." It is a voluntary muscle which has to do with the completion of the act of defecation. It aids in the final expulsion of the terminal portion of the stool in a clean manner. In the case of diarrhea or an unusual accumulation of gas it is valuable as a voluntary *accessory* sphincter, and thus has a high degree of social importance. If it is severed, the process of cleansing after defecation is much more difficult and washing is often required. For these reasons if the external sphincter can be spared it is advisable to do so, although most patients can carry on in a highly satisfactory manner even though it is divided. The division of the internal sphincteric ring is not necessary in cases of anal fistula, although it may be in the more complicated and deeply seated fistulas.

Whether or not the infectious process reaches the ischiorectal fossa between the sphincters or more superficially around the external sphincter, depends in many instances upon anatomical variations in the anal region. It is recalled that the anal canal begins as a dimple in the ectoderm which grows inward to meet the endodermal hind gut. Fusion of these organs occurs about the eighth week of embryonic life. At the point of fusion is the pectinate line, the juncture of the squamous epithelium of the anal canal and the columnar epithelium of the rectum. It is at this point that the pouches of the crypts of Morgagni extend downward underneath the upper portion of the anal canal. The length of the average anal canal is one and three-tenths inches, and its entire length is lined with squamous epithelium. Anal canals vary in length from one-half to two inches, so that in some cases the rectal epithelium may extend downward through the canal almost to the anus. In this case the pectinate line is almost at the anal orifice and the squamous epithelium extends upward only a small fraction of an inch. In these individuals when the margins of the anus are slightly retracted, the red velvety rectal mucosa is plainly visible. This means that the pockets at the lower end of the crypts of Morgagni are situated low in the anal canal just above the anal orifice. (Fig. 1, Part II.) It is evident that infection occurring in a crypt in this position would produce a superficial abscess and one which would not likely involve the external sphincter muscle.

There is also considerable variation in the sphincteric musculatures, which also has to do with the location of abscesses and fistulas occurring in this region. The first is the relationship of the internal and external sphincteric rings. (Fig. 1, Part I.) These bands may be quite closely incorporated together. (Fig. 1, Part IA.) They are always distinct upon dissection, but by palpation it is often difficult to feel a line of demarcation between them. In other individuals the rings are quite far apart. (Fig. 1, Part IB.) A very definite space or depression can be felt between them and each can be palpated as a separate entity. When the two bands are closely incorporated, it is evident that an infection would be less

likely to extend between them. Finally, there is a great variation in the relative position of these muscles to the anal canal and crypts of Morgagni. In some cases the external sphincter (occasionally also the internal) surround the anal canal at its very lower-most end. In fact, the muscle stands out as a cord-like band plainly bulging through the perianal skin external to the anus. In other individuals nothing can be seen externally and the muscle is not visible through the skin, even though it is contracted voluntarily. Palpation will reveal the muscle deeply placed and surrounding the anal canal high above the anal orifice.

If we take two extremes as an example, it is perfectly evident what will occur. If the pockets of the crypts of Morgagni are situated at the upper margin of a long anal canal, and the sphincter muscles surround the canal near the anal orifice, infections arising in such crypts are well above the sphincter muscles. As the infectious process burrows downward it is likely to spread laterally through the space between the sphincters. This is almost certain to occur if the sphincteric rings are rather widely separated, less likely if they are closely approximated. If the reverse is true, i.e., the sphincter is situated high and the crypts low in the anal canal, infection occurring in such crypts is actually below the sphincter and hence the infection will spread laterally without involving the muscle. (Fig. 1, Part II.) Between these

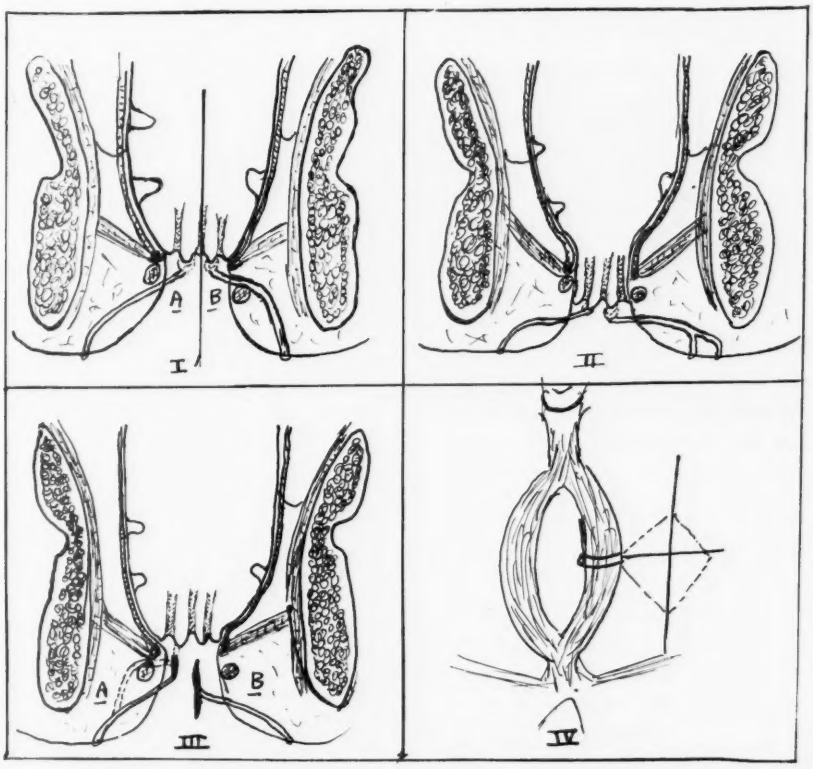

Fig. 1. Diagram of anatomical variations which often influence the involvement or noninvolvement of the external sphincter in anal fistulas; proper method of draining anal abscess. I, termination of crypts cephalad to sphincteric rings: (A) sphincteric rings closely approximated; (B) sphincteric rings widely separated. II, termination of crypts caudad to sphincteric rings. Showing closely approximated and widely separated types. III, fissure-in-ano: (A) fissure arising from crypt; may cause fistula involving sphincter; (B) fissure usually arising from trauma near the anal margin; almost never causes fistula involving sphincter. IV, proper method of draining perianal abscess. Dotted lines indicate amount of skin and overlying fat to excise to prevent too rapid closure of wound.

two extremes lie the anatomical variations which influence the manner in which infectious processes extend from the crypts of Morgagni to the ischiorectal fossa. These anatomical variations, plus tension determines whether the infection spreads above or below the external sphincter, which in turn, determines whether or not external sphincter must be divided in operating upon an anal abscess or fistula. In special instances, other factors may have a bearing upon the manner of the spread of the infection, but in general we believe these to be the most important considerations of a general character.

The location of the infected crypt usually has a direct bearing upon the location of the subsequent abscess. In the case of infection of lateral crypts, the abscess is most likely to form directly in the adjacent ischiorectal fossa. If the posterior crypt is involved the abscess usually forms in the midline posteriorly, or a little to the right or left of the midline. Anteriorly it may be either in the midline or slightly lateral. Fat has little resistance to infection, and once the infection has reached the ischiorectal fossa, there is a further tendency for this process to spread. This extension may simply be through the fat of the one fossa, or it may extend posteriorly underneath the anococcygeal tendon and involve the adjacent fossa. This same tendency to spread may occur anteriorly, and in this location, extension is seen more often in the female than in the male. Anteriorly, the extension is usually along the transversus perinei muscle. Almost invariably the extension of the process ceases the moment the abscess is incised and tension relieved. For this reason, contrary to the belief of many, we believe that the earlier these abscesses are drained, the better. In our opinion it is a mistake to treat the condition expectantly and wait for the abscess to "point." To do so only invites the formation of a larger abscess cavity, the extension to more distant points and greater tissue destruction. It has been our custom for the past twenty years to drain these abscesses the moment we could be sure that pus was present, even though there is no external redness of the skin or other superficial evidence of abscess formation. We have had no occasion to regret it. In most instances in which we have been confronted with extensive and complicated fistulas, the patients have failed to see a physician and the abscesses finally ruptured, or they were treated by some physician with hot packs and Sitz baths, rather than immediate drainage.

When an abscess is seen, it is always a question as to whether or not a primary or internal opening is present, although it should be assumed that one is present until proved otherwise. This can often be determined by dilating the anal canal and lower rectum with a bivalve speculum, and carefully examining the rectal crypts with a blunt hook. Often the tip of a hook can be passed through the opening in the infected crypt into the abscess cavity, or pus can be seen oozing into the crypt from the abscess. If the opening is not found in this manner, the abscess is opened and gentle probing of the cavity may reveal an opening into a crypt. In both instances, examination should be done carefully and gently, so no opening is made where none existed. If no opening is found, the surgeon should be content with draining the abscess. If an opening is found, it is a question of whether or not the intervening tissue should be divided and the entire operation completed, or whether the abscess should be drained and the fistula operated upon at a later date.

Our course is determined by the following factors: In all cases in which the tract lies below the external sphincter, we complete the operation without delay. If the abscess is small, even though the sinus is above the external sphincter, we also complete the operation immediately. On the other hand, if the abscess is large and for this reason the margins of the wound gape widely after incision, thus widely retracting the cut margins of the sphincter, we simply drain the abscess. Later after

the cavity has contracted, the sphincter is divided. The smaller cavity retains the cut margins in much closer apposition, and we believe there is less final deformity than if the operation is completed when the abscess was drained. All large abscesses should be widely opened by a crucial incision, and one limb of the cross should be carried down to the outer margin of the sphincter (Fig. 1, Part IV.) It is our custom in the case of large abscesses to remove a portion of the overlying skin which prevents too rapid healing of the incision and insures adequate drainage without daily packing. By carrying one arm of the incision to the outer margin of the sphincter, only a short tract passing under the sphincter to the crypt is left to be divided subsequently. A seton is passed through this tract and tied around the sphincter. This keeps the tract open and acts as a guide at the time the muscle is divided. This final step of the operation is so simple that it usually can be done in the physician's office with the aid of a small amount of local anesthesia.

In many instances the physician does not see the patient in the abscess stage but only after the fistula is present. Sometimes this is many years after formation of the original abscess. Buie states that 69 per cent of his patients had had their fistula more than one year and 22 per cent for more than nine years. In cases of long duration, there is ordinarily a history of repeated attacks. The usual story is that the fistula heals externally and discharge ceases. Then after varying periods of time, some tenderness and induration is noted and there is a return of the discharge. This process may be repeated many times. In most instances the recurrence is in the same location and the discharge is at the site of the original opening. In other cases the infection breaks through the walls of the original cavity and rupture occurs externally at a different site. The number of external openings which may be developed in this manner is almost unlimited. One of our patients had forty-two external openings. Regardless of the number of external openings, there is usually but one internal or primary opening. The external openings are merely caused by burrowings of the infectious process. All connect at some point with the original sinus. While anal fistulas almost invariably have a single internal opening, multiple internal openings are often present in fistulas arising higher in the rectum. These fistulas usually result from infectious processes above strictures which may occur from any inflammatory process, but are frequently seen in the female following lymphopathia veneria infection. They also occur in cases of actinomycosis, advanced carcinoma, or occasionally in tuberculosis.

While in anal fistula only one internal opening is usually present, the ramification and extent of the tract (or tracts) before the surface is reached is unpredictable from external appearances. In some instances the extent cannot be determined by preoperative examination. According to Salmon, if a transverse line is drawn across the anus midway anteriorly-posteriorly, any fistula whose external opening is posterior to this transverse line will have its primary opening in the midline posteriorly. Any fistula having its external opening anterior to this line, will have its internal opening directly into the nearest crypt. This is not an invariable rule but it is usually correct and gives a good clue as to where the internal or primary opening may be.

The primary requisite for the successful operation for fistula is that no tracts or branches of the fistula be overlooked at the time of operation. To prevent this, several methods are advocated to determine the extent and location of the tracts before operation is begun, and also to aid in following the tracts at the time of operation. The two most widely used are the injection of bismuth paste, or soda iodide solution, followed by *x*-ray and the injection of various dyes. In anal fistula neither of these methods has been of any particular help to us. If there is a single tract, it is easily followed; and if there are lateral

branches present, they are frequently not penetrated by the bismuth or sodium iodide, and hence the surgeon may be confused rather than helped by the *x*-ray plates. We do find the injection of bismuth or sodium iodide to be of help where the site of the origin of the fistula is in some distanct place, as in a sigmoid diverticulum, or where there is a connection with the bladder or other viscus. Various dyes are also injected in order to stain the tract, determine the site of the internal opening and enable the surgeon to follow the tract more readily at time of operation. Again we think this unnecessary, as anyone familiar with this type of surgery should be able to follow the tracts by watching the granulation tissue lining the sinuses and carefully probing and palpating the tissues. The injection of any substances into the fistulous tract is not without danger of perforation of the tract and spread of the infection. The injection of dyes is also open to the same objection as the use of bismuth and sodium iodide, i.e., all accessory tracts are not penetrated. If a fistulous tract is injected, it should be done gently and without pressure. Hydrogen peroxide or other gas-forming solutions should never be added to the dyes. There are several methods whereby the surgeon can gain some preliminary knowledge as to the nature of a fistula. If possible, the location of the internal and external openings should be determined. The external opening or openings are usually apparent. If the anal canal and lower portion of the rectum can be dilated with a bivalve speculum, inspection of the crypts and anal canal will often reveal the internal opening, or it can be found with a crypt hook. Slotted anoscopes are less satisfactory and in some instances of anal stenosis or painful anal lesions, inspection must be deferred until anesthesia is available.

Palpation is often of great value. If the index finger be inserted into the rectum and the adjacent structures palpated between the thumb and finger tip, a cord-like tract can often be felt extending from the external opening to the base of a crypt. If this is not possible, the palpation of the cryptic area alone, will often reveal a small hard area indicating which crypt is affected. In other instances a rather bulky area of induration is felt adjacent to the anus. This often indicates the general direction of the tract and is suggestive of accessory sinuses being present.

The fistula is now examined with a fine probe. An ordinary malleable probe is usually useless because of its diameter. We have found the Early probe, which has both a straight and a curved end and is made of fine-gauge stiff wire, most satisfactory for this purpose. In many instances of simple fistula, the probe can be passed entirely through the tract and into the bowel. Regardless of the method of examination, it may be found at operation that a seemingly simple fistula will have one or more accessory branches which are far more extensive than the tract which was discovered at the time of examination. Beginning an operation for fistula with too definitely preconceived indeas as to extent, is likely to result in some accessory sinuses being overlooked. One reason we object to the use of bismuth and dyes is that they may give the surgeon a false sense of security as to his knowledge of the extent of the lesion.

The cure of a fistula is based upon a very few fundamental "musts." The internal opening must be found. An incision must be made from the external to the internal opening. All accessory sinuses must be found and either incised and curetted or dissected out. At the completion of the operation, a broad, clean, flat wound without overhanging edges must be created.

The first step in the operation is to locate, if possible, the internal opening. Under anesthesia this is often possible, where before the attempt has been unsuccessful. The anus and lower rectum are dilated and the crypts examined as previously described. If the opening is not found, the operator should attempt to manipulate the probe carefully through the

tract by inserting the tip into the external opening and gently working toward the bowel. In many instances in which the tract is tortuous, it will be impossible to pass the probe through the entire tract to the primary opening. In this case it is best to insert the probe as far as possible without undue force and then incise the sinus as far as the probe has been inserted. Here the tract will often be found to have made a turn at an angle so acute as to have prevented further passage of the probe. However, with the distal portion of the tract incised, the probe can now be further advanced along the sinus. Proceeding in this manner, each time incising that portion of the sinus through which the probe has been advanced, the operator arrives at the internal opening of the fistula. It is well to follow the tract carefully and to disturb it as little as possible until the internal opening is located. The removal of lateral tracts or a portion of the main tract before the primary opening is found, often renders its discovery much more difficult. In cases in which the internal opening is difficult to locate, a maneuver of Newton Smith is often of value. After the fistulous tract has been partially freed, tension is made on the scar tissue, carefully watching the cryptic area. In many cases the traction will cause a dimpling in the base of the crypt where the fistula originates. In the rare instances in which the internal opening has healed over the surgeon can still be sure of the site of origin.

After the primary sinus has been incised, all secondary branches must be located and either incised or excised. As the first step toward this end, the granulation tissue lining the primary sinus should be carefully and thoroughly curetted away so the smooth and glistening scar tissue forming the tract is plainly visible. When this has been done, the openings to the accessory sinuses can usually be recognized by small tufts of granulation tissue extruding into the parent sinus. Curetting of the main sinuses will not remove these tufts and they stand out in distinct contrast to the smooth fibrous tissue walls of the primary sinus. A probe is now inserted into one of these accessory sinuses and the tract incised. After it has been incised to its termination, it is curetted or removed as is thought best. Proceeding in this manner all accessory tracts are dealt with in a similar way. All tissues in the suspected area are carefully palpated between the thumb and index finger for any indurated areas. This may reveal a further accessory sinus which has been previously overlooked. It is very easy to miss accessory sinuses and it is important not to be hurried, but to be careful and deliberate in searching for them. As has been said, no one can be certain before the operation, just how extensive a fistula may be. Lack of recognition of this fact, coupled with being too hurried, is a material factor in the frequent unsuccessful operation for fistula. Contrary to some, we do not believe it advisable to dissect out all of the fistulous tract. In no event should that portion of the tract containing the internal opening be removed. To do so causes a greater gaping of the terminal opening of the rectum, a greater subsequent deformity and a greater loss of sphincteric function. This portion of the tract should never be treated other than by simple incision. Probably the second most frequent cause of failure, is the failure to be radical enough in the operative procedure. At the completion of all fistula operations the site should present a broad flat, clean wound with no overhanging edges. To accomplish this the skin margins should be trimmed back widely and the underlying fat removed. All ragged tissue in the wound, resulting from the incision of the fistulous tracts should be excised. This type of wound will heal readily and completely, and after-care is simple and painless. Where a deep narrow wound is left it must be packed daily and the dressing is painful. Bridging of the wound is more likely to occur, and in any event the packing will produce a greater amount of scar and subsequent deformity. A broad, flat wound simply requires daily cleansing which is

painless and no packing is necessary. Occasionally, at the completion of the operation a small gauze wick is placed in the wound, but once this is removed it is not replaced.

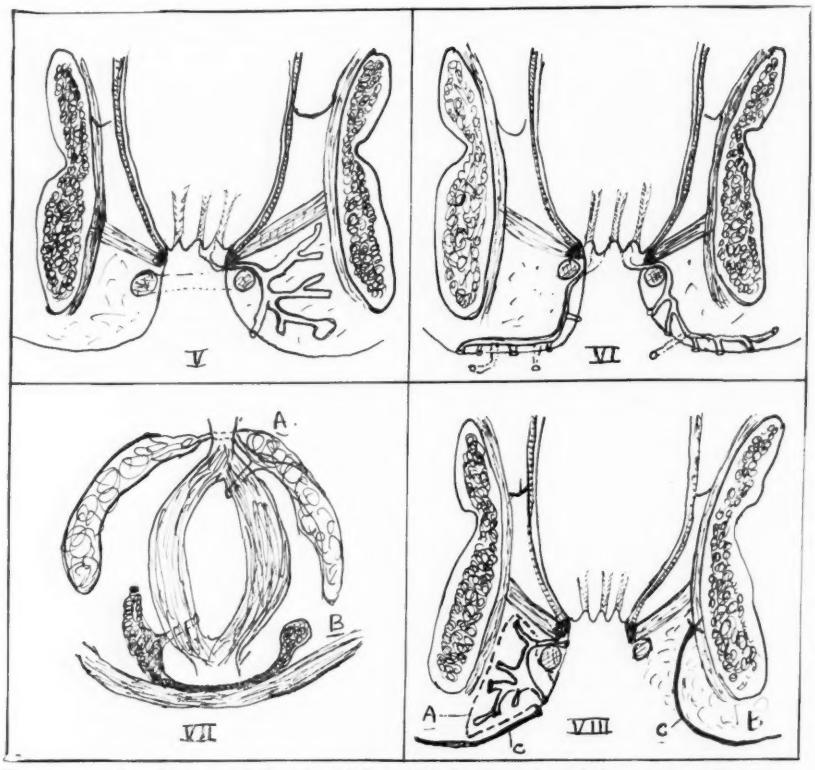

Fig. 2. v, deeply seated fistula with multiple sinus involving one entire fossa; usually one (or more) external openings rather near anus. vi, superficial fistula with many external openings far from the anus; deeper portion of ischiorectal fossa not involved. vii, posterior and anterior horseshoe fistulas indicating most frequent method of spread: (a) posterior—behind the anococcygeal tendon; (b) anterior—along the sheath of the transversus perinei muscle. viii, diagram of operation for deeply seated fistula shown in v: (a) block of tissue to be removed outlined—and skin to be saved (c); (b) skin flap (c) sutured in place to cover defect partially; external sphincter divided.

Anal fistulas which have more than a single tract, usually result from failure to drain the abscess early. These fistulas may be classified in three general groups: First, fistulas in which the secondary tracts are superficial. (Fig. 2, Part vi.) In these fistulas, the accessory tracts are caused by the infectious process burrowing in the superficial fat adjacent to the anus and over the buttocks. These fistulas may have numerous openings at great distances from the anus. Because of the length of the tracts, they present a rather serious appearance but the fact that the tracts are superficial, renders their treatment more simple than those, in which external appearances may not indicate so extensive a lesion. The long superficial tracts should be incised down to the original sinus. Only a single sinus usually connects with the rectum. When this is incised through to the internal opening, healing usually occurs promptly and with no more deformity or loss of function than occurs in the case of a simple fistula consisting of a single tract.

In the second group, the secondary tracts are located in one ischiorectal fossa but are deeply placed and the fossa may be honeycombed with these accessory sinuses. (Fig. 2, Part v.) There is usually but a single external opening, and until the operation is under way the surgeon may believe himself to be dealing with a simple fistula consisting of a single tract. In these

cases it is often necessary to dissect out the entire contents of the ischiorectal fossa. This leaves a deep cavity with its apex above the internal sphincteric ring. A wide area of skin and fat of the ischiorectal fossa should be removed, leaving a cone-like wound with the base external. This type of wound will heal readily without bridging. A wound left by a less radical operation is almost certain to bridge over and require subsequent operation. In very extensive cases a skin flap may be left to cover the defect partially. (Fig. 2, Part VIII.)

The third type of fistula is the so-called horseshoe fistula, the fistulous tract extending around the anal canal either anteriorly or posteriorly in the shape of a horseshoe. (Fig. 2, Part VII.) This condition results from an abscess which originates in one fossa and then extends to the other. This condition more than any other offers the most conclusive proof of the advantage of early drainage of perianal abscesses, as opposed to the method of procrastination and "expectant" treatment. If one has had the opportunity to watch the formation of one of these fistulas, the sequence of events cannot be misconstrued by the most casual observer. There is first an abscess in one ischiorectal fossa, and it is only after the abscess has been present for some time and the pus is under tension, that it will be seen to spread into the adjacent fossa. Early incision would of course have prevented this.

In the case of a posterior horseshoe fistula the spread is usually through the space just posterior to the anococcygeal tendon. Incision of the tract should be made from end to end of the horseshoe. The anococcygeal tendon is best divided. This will cause an anterior and upward retraction of the anus which causes some deformity, but does not cause any impairment of function. In most cases the primary opening will be found in the midline posteriorly. We usually do this operation in two stages, since we believe this method causes less residual deformity. After the tract is incised and the margins trimmed back, a seton is passed through the internal opening and tied posteriorly. This bridge of tissue is divided after the remainder of the tract is contracted and partically healed. An anterior horseshoe fistula occurs less frequently and is more often seen in women. The arms of the horseshoe are not likely to be as long as in the posterior type. Again we prefer to do this operation in two stages. In the female there is likely to be considerable deformity and some difficulty in accessory bowel control. It has been definitely noted that division of the external sphincter anteriorly causes a wider gaping, a greater impairment of function than division elsewhere.

Operation for simple fistulas consisting of a single tract is minor surgery, but operation upon complicated fistulas is distinctly major surgery. Failure to recognize this fact is the chief cause for the repeated operative procedures and miserable results in many of these cases. There is no rule of thumb by which these patients can be operated upon, and that is the rub for the average surgeon. Every complicated fistula must be viewed as a new problem which is likely to present certain conditions not encountered before. All cases should be approached with some diffidence. A thorough knowledge of the anatomy of the region is essential. More important is a knowledge of what occurs in the way of deformity and impairment of function when the various muscles, tendons, nerves and other tissues of this region are cut, injured or removed. These last facts I am afraid are not too common knowledge, and without this knowledge complicated fistulas cannot be cared for to the best advantage.

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ANAL, PERIANAL, PERINEAL AND SACROCOCCYGEAL SINUSES*

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THE term "sinus," in a pathologic sense, pertains to a tract or tube, inflammatory in origin, blind at one end and communicating at the other with a cutaneous or mucosal surface.

"Fistula" is the Latin word for "pipe" or "reed," and pertains to a tract or tube, inflammatory in origin, and open at both ends. Both openings may lie upon the internal surfaces of hollow viscera, or one may lie internally and the other may involve an external surface of the body. Hence a fistula can be described as an abnormal communication between two hollow organs (e.g., rectovesical fistula) or as a communication between a hollow viscus and a body surface (e.g., fecal fistula).

Accordingly, therefore, the terms sinus and fistula are not synonymous. Failure to make this distinction is responsible for considerable confusion relative to pathologic processes in which fistulas and sinuses are formed.

Perhaps the safest plan in view of the subjects at hand is to consider all openings in the anal and perianal skin, the skin of the perineum and the skin of the sacrococcygeal region, from which there is any kind of discharge, as draining "sinuses." Steps must then be taken to prove that a given draining sinus is actually a sinus or that it is one opening of a true fistula according to the premises stated above.

N. D. Smith[19] has provided a most practical classification which, in the essayist's humble opinion, serves admirably to indicate the ramifications of the problem of sinuses in the region of the anal outlet:

"Group I. Spontaneous inflammatory processes:
1. Fistula-in-ano
2. Incomplete fistula
3. Sinus or fistula associated with an anal fissure
4. Rectovaginal fistula
5. Infection of lymphatic vessels
6. Periurethral abscess

"Group II. Lesions arising in glandular structures or cysts:
1. Pilonidal cysts
2. Chromaffin bodies
3. Multilocular cysts
4. Bartholin cyst abscesses
5. Pyodermia (hidradenitis suppurativa)
6. Dermoid cysts

"Group III. Lesions due to unusual etiology or direct trauma:
1. Tuberculosis
2. Actinomycosis
3. Osteomyelitis
4. Penetrating injuries
5. Lymphogranuloma inguinale
6. Malignant lesions such as carcinoma, epithelioma and lymphosarcoma

"Group IV. Pseudosinuses:
1. Congenital anal dimples
2. Post-anal dimple
3. Comedones
4. Marked enlargement of hair follicles."

The author will attempt to consider first those sinuses occurring in the anal, perianal and perineal regions, and lastly, sacrococcygeal sinuses. It is acknowledged that a pilonidal sinus may present at a point lateral to the anus and even anterior to it,

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but for practical purposes this plan should serve adequately.

ANAL, PERIANAL AND PERINEAL SINUSES

Inasmuch as the majority of so-called draining sinuses in the anal, perianal and perineal regions actually represent the secondary openings of anal fistulas, a consideration of the pathogenesis of anal fistula is indicated at once. Confusion with regard to this subject is inevitable in view of the teachings that an anal or ischiorectal abscess must be incised promptly or else a fistula will result, that an abscess is the first step in the formation of a fistula, and that most anal fistulas are tuberculous in origin.

It is not uncommon to find, in most textbooks of anorectal disease, that abscesses are discussed in one chapter and fistulas in another. The student is thus led to believe that the two processes have very little, if any, relationship to each other. An attempt will be made in the next few paragraphs to show, according to the teaching of Buie,[5] that abscess formation is merely one stage in the development of an anal fistula. A thorough understanding of these processes is essential to proper diagnosis and to adequate therapy and will serve to dispel unwarranted confusion relative to the subject.

Figure 1, part 1, is a diagrammatic representation of a longitudinal section through the anal wall, the wall of the distal portion of the rectum and the anorectal junction. At the bottom of the anal crypt there is usually a well marked transition from rectal mucous membrane to anal skin. Also in or near its base the anal crypt communicates with one or more anal ducts which lead to vestigial anal glands. The nervous physiology of the anorectum is such that the skin of the anal canal (including the structures of the dentate margin) and the perianal skin are supplied by somatic sensory nerves. The rectal mucosa harbors, if any at all, only visceral sensory fibers. It follows, therefore, that rectal lesions, except those which lie close to the dentate margin, do not give rise to pain early in their course. Anal and perianal lesions on the contrary are commonly featured by varying degrees of pain or discomfort.

The story of the anal ducts and anal glands is interesting in view of the attention which has been accorded them again in recent years. Herrmann and Desfosses[10] described these structures in 1880 and said (author's translation): "From a surgical standpoint these long tortuous ducts, marked at their extremities by small follicles, present a certain interest particularly with regard to the role which they can play in the production of complicated fistulas which one encounters so frequently in this region."

In 1914, F. P. Johnson[11] published his splendid work on the development of the rectum in the human embryo including the embryology of the anal glands. He called attention to the work of Herrmann.

Pennington,[16] in 1917, wrote: "That an important anatomic fact could in this scientific age, be disregarded or lost sight of for many years, after being placed on record, and then, without knowledge of its former discovery, be rediscovered and found to be of great importance as a disease producing factor, is almost unbelievable. However, this anatomic finding, when resurrected, proves to be a vital etiologic factor in the causation of fistula and other diseases of the rectum, with their metastatic sequelae."

In 1933, Tucker and Hellwig[27] presented their work on the histopathology of the anal crypts, including an excellent comparative anatomic study, before the American Proctologic Society. Pope and Gunn, in discussing this paper, presented their own work which confirmed the findings of Tucker and Hellwig. These authors made no mention of the writings of Herrmann or of F. P. Johnson.

It is thought that infectious material from the stools, with or without trauma, frequently collects or is caught in an anal

crypt. The associated anal ducts easily afford portals through which infection can gain entry into the perianal tissues. (Fig. 1, parts 2 and 3.) The involved crypt thus becomes the "primary" opening of the fistulous tract which as yet has not been completed.

Then follows the second step—that of invasion of the soft tissues via the lymphatics and blood vessels, the lymphatics probably playing the more important rôle. (Fig. 1, part 3.)

Once infectious material has invaded the soft tissues of the ischioanal fossa the stage is set for abscess formation. (Fig. 1, part 4.) It is during this third stage, and not before, that the patient usually presents himself to his doctor with all the signs and symptoms of acute inflammation: redness, swelling, local heat, tenderness and pain. A practical point is well worth considering here. An anal abscess (frequently called ischiorectal or ischioanal abscess) should be incised as near the anal margin as possible without disregarding the point of fluctuation if present. Thus the doctor may be able to establish a shorter fistulous tract.

The fourth and final stage in the development of an anal fistula pertains either to spontaneous rupture of an anal abscess or to incision and drainage of the abscess. Either occurrence provides the other or "secondary" opening, thus completing the fistula according to the definition set forth above. (Fig. 1, part 5.) The patient is much relieved when his abscess is drained, and the abscess cavity rapidly contracts to a narrow tube. (Fig. 1, part 6.) Thus it can be said that an anal fistula is, in effect, a chronic anal abscess. It is well, however, for the doctor to explain to his patient that the relief which he has obtained following drainage of the abscess does not mean that his trouble has been cured. A cure cannot be accomplished without the performance of a "fistula operation." The author always attempts to explain the problem to his patient by means of simple diagrams similar to those shown in Figure 1.

The terms "primary" and "secondary" have been suggested by Buie[6] for the openings of an anal fistula in order to correct the confusing terminology which has heretofore been applied to these openings. The point

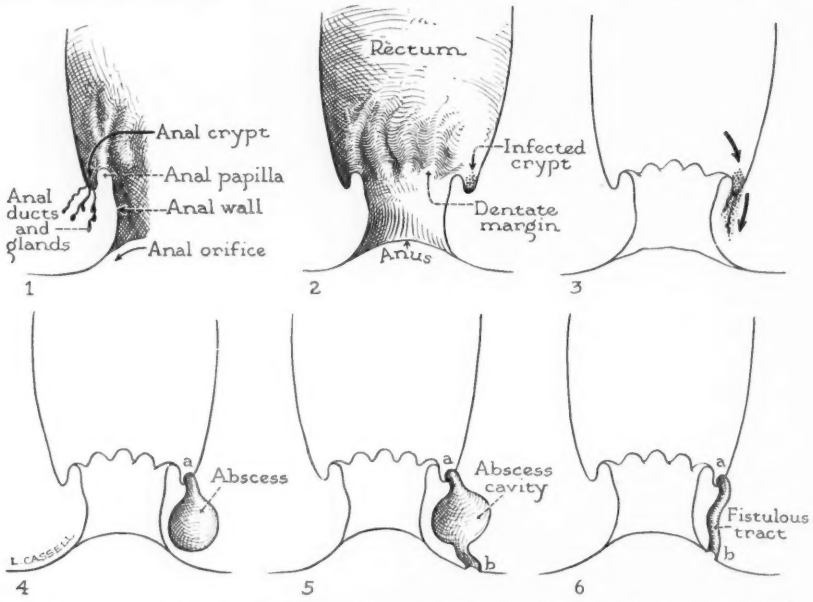

Fig. 1. 1, coronal section illustrating anal crypt, and anal ducts and glands. 2, coronal section; first stage of formation of anal fistula. 3, coronal section; second stage of formation of anal fistula. 4, coronal section; third stage of formation of anal fistula. 5, coronal section; fourth stage showing completion of fistula: (*a*) primary opening; (*b*) secondary opening. 6, coronal section; chronic anal fistula: (*a*) primary opening; (*b*) secondary opening.

of origin of an anal fistula, which has been shown to be an anal crypt, is commonly known as the "internal" opening. The terminal point is designated as the "external" opening. These terms are entirely proper for an anal fistula which ends in anal, perianal or perineal skin. When, however, the terminal opening lies in the rectal wall above the dentate margin, or in the vagina, etc., that opening, too, becomes "internal," and confusion is inevitable. If the involved anal crypt be known as the "primary" opening, then any and all other openings associated with this same fistula can be termed "secondary." The author heartily endorses this suggested terminology and finds that his students more readily grasp the relationship between pathogenesis and terminology with regard to the subject of anal fistula.

With regard to the pathogenesis of anal fistula some further elaboration must be considered relative to the "surgical spaces" about the anorectum. We shall then be prepared to take up the important subject of diagnosis—the theme of this symposium.

A surgical space actually exists only when the tissues normally present there are destroyed or displaced by the products of inflammation, i.e., abscess formation. These spaces can be created for demonstration in the cadaver by means of dissection. They are five in number.

Reference to Figure 2, part 1 calls our attention to two ischioanal spaces below the levators and to two pelvirectal spaces above the levators. There is a third space above the levators known as the retrorectal space which is best shown in sagittal and cross sections. However, for the purposes of this paper we shall consider only the two infralevator spaces known more commonly as the ischiorectal spaces or fossae. The author prefers the term "ischioanal" suggested by Batson[2] in view of the fact that the levator ani muscle with its investing fasciae (pelvic diaphragm) passes between the internal and external sphincter muscles to gain attachment to the wall of the anal canal. Thus this space is more closely related on its medial side to the anal canal than to the rectum, hence Batson's term "ischioanal."

The ischioanal fossa is involved, usually superficially but sometimes deeply, in the third and fourth stages of the development of an anal fistula. When a more complicated fistula, such as the "horseshoe" type, is present both ischioanal spaces are involved.

For all practical purposes the diagnosis of anal fistula depends on a history plus the findings afforded by two well known procedures in physical diagnosis—inspection and palpation. The facts thus obtained, and evaluated in the light of the pathogenesis of anal fistula set forth above, should enable the careful physician to arrive at a proper diagnosis.

The patient can usually remember the first abscess, and whether it ruptured spontaneously or had to be "lanced." Even if he fails to remember the abscess he will report the characteristic periodic drainage from a "point near the rectum" (meaning anus, of course). The patient may report having had several abscesses. This most likely has been a recurrence, at irregular intervals, of the same trouble. As long as a fairly constant drainage occurs there will be no recurrence of the abscess. When, however, the secondary opening becomes "healed over" there may result a backing up of secretions and exudate with reappearance of the signs and symptoms of acute inflammation. Unless the doctor understands the nature of an anal fistula he may think that he is dealing with a new abscess each time. As noted previously the author endeavors to explain to his patient, as simply as possible, that drainage of the abscess merely affords relief from the acute symptoms, and that more radical surgery (fistulectomy) is essential to a cure.

With regard to inspection let us consider first external inspection, and later on, internal inspection or endoscopic examination.

The examiner closely scrutinizes the skin of the buttocks, lower back and perineum. Then, upon gently retracting the buttocks, he can see the medial aspects of the but-

tocks and the anal and perianal skin. A draining sinus (to be considered the secondary opening of an anal fistula until proved otherwise) may present as a small elevated opening with some discoloration of the surrounding skin. Granulation tissue is likely to border on the edges of the opening. In the instance of a secondary opening of long standing there may be considerable scar tissue present. The opening may even appear to be "healed over." There may be one or several draining sinuses. The approximate location of each opening and its distance from the anal margin should be noted. The amount of discharge from a draining sinus varies greatly. When there

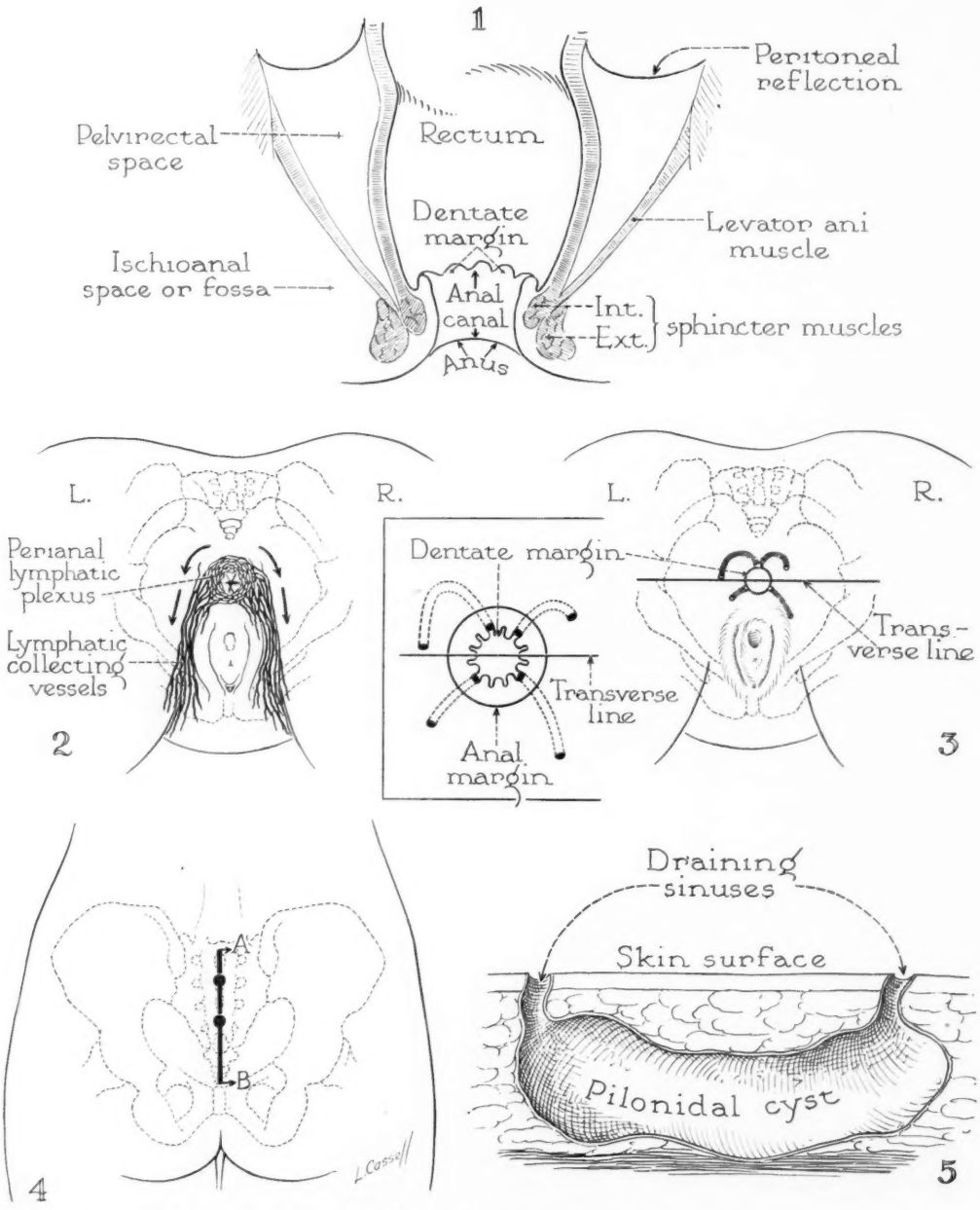

Fig. 2. 1, coronal section illustrating the surgical spaces about the anorectum (except the retrorectal space). 2, anal and perianal lymphatics in the female. 3 and inset, illustrating Goodsall's rule. Modified from Gabriel. 4, posterior view showing sacrococcygeal region. The line, A–B, represents the usual sites of pilonidal sinus openings in the skin of this region. 5, longitudinal section along the line A–B of 4.

has been copious discharge over a considerable period of time, the patient is likely to have resorted to the constant use of some kind of dressing.

Gentle palpation of the tissues about the draining orifice may disclose a firm, subcutaneous "cord" leading from the opening toward the wall of the anal canal. This represents the fistulous tract itself connecting the primary and secondary openings. (Fig. 1, part 6.) Whether or not a tract can be felt on superficial palpation the examiner should next proceed to a careful "bidigital" examination of the anal and perianal tissues. This is done by inserting the examining finger (index) within the anal canal and lower rectum. One can then palpate the tissues in question between the index finger and the thumb of the same hand. Such an examination frequently yields information that would have been overlooked ordinarily. This procedure is especially valuable at the time of surgery when complete relaxation of the anal outlet has been established by means of regional anesthesia. Under these circumstances the author frequently discovers a fistulous process of which he was unaware at the time of the first examination of the patient.

In view of the stages of development of an anal fistula as outlined previously it is readily apparent that the doctor must carefully examine the structures comprising the dentate margin and forming the superior boundary of the anal canal, the inferior boundary being the anal margin or anal verge. Careful palpation may enable the examining finger to follow the cord or tract directly to the primary opening in an anal crypt. If the examiner cannot identify a tract, he may be able to feel induration or scarring in the region of the involved anal crypt. Here, again, bidigital examination is an invaluable procedure. The examiner may find an area of induration above the dentate margin beneath the rectal mucosa leading to a "puckered" area in the mucosa. This is most likely an "internal" limb of an anal fistula, the secondary opening of which lies in the mucous membrane of the rectum. This type of fistula is not, however, within the scope of this paper.

Following external inspection, and palpation of the anal and perianal tissues, the doctor next proceeds to internal inspection or endoscopic examination by means of an anoscope or a proctoscope. This procedure should serve to confirm or to disprove the impressions which the examiner has obtained on palpation. The principal goal here is direct inspection of the dentate margin. Not infrequently the involved crypt can be identified by means of a drop or two of pus seen oozing from it. Again the scarring in the region of the involved crypt may have caused sufficient deformity to be recognized on direct inspection. Finally, it may be that direct inspection of the dentate margin will yield no evidence whatsoever of a primary opening. The examining finger may have disclosed more information than the anoscope.

At this point the reader's attention is called to the fact that no mention has been made thus far of the use of probes or of the use of dyes in attempting to determine the course of a fistulous tract.

The author is heartily in accord with the teaching of Buie and N. D. Smith relative to the use of probes. Nearly every new patient who consults the proctologist is in fear of the examination because of unsavory stories which he has heard about proctoscopy or because of a previous unfortunate experience in the hands of another doctor. Since there is absolutely nothing to be gained by causing a patient to suffer excruciating pain at the time of his examination, and in view of the fact that sinuses and fistulous tracts can be explored by the use of probes with considerably more satisfaction when proper anesthesia has been established, it seems only logical to defer "probing" until the time of surgery. The author does not hesitate to suggest to a patient who has an extremely painful condition that he submit to an examination under regional anesthesia in the operating room where the investigation can be done without causing pain and

where surgical treatment can be carried out as indicated at the time. The suffering patient is usually quite willing and the advantages of such an arrangement to the doctor are obvious.

Although the details of the surgical treatment of anal fistula are not within the scope of this essay, a point of practical importance relative to the use of probes at the time of surgery seems in order at this point. With the anal canal adequately dilated a soft, silver probe should be bent at a fairly sharp angle and gently introduced into the suspected anal crypt. If the tip of the probe can be recovered at the secondary opening of the fistula, the tract has been explored from its primary opening or point of origin to its secondary opening or point of termination. This procedure helps to "drive home" our conception of the development of an anal fistula. When, however, one fails to pass the probe in the manner just described, he can then straighten the probe and attempt to explore the tract from its secondary opening, thus hoping to recover the tip of the probe at the primary source of the fistula. Even this method may fail, usually because of a sharp angulation in the course of the tract. Should this occur the surgeon can pass one probe through the secondary opening and another probe through the suspected primary opening, and he can usually determine by feel when the tips of the probes come into contact.

It is hoped that the description of the pathogenesis of an anal fistula which has been given in the foregoing pages will serve to indicate to the doctor that dyes, colored solutions, etc., are not essential to the discovery of a primary opening. He should know that the point of origin of the fistula is to be found in the dentate margin. Nor is the staining of a fistulous tract by some kind of dye (e.g., methylene blue) necessary for its identification. The characteristic appearance of the wall of the tract, when laid open, is discernible grossly. The author's preceptor, the late Collier F. Martin, was of the opinion that the surgeon who used a dye in attempting to demonstrate an anal fistula was usually successful only in demonstrating his lack of knowledge of the pathogenesis of this entity.[12]

In the occasional instances of a sinus resulting from an impalement injury or of a sinus which is osteomyelitic in origin the doctor is justified in the use of injections of lipiodol to facilitate roentgenologic study of the location and extent of the sinus. He should remember, however, that the injection mass may follow normal planes of cleavage and thus misrepresent the true state of affairs.

A question might be asked logically at this point regarding the location and pathways of anal fistulas. Do anal fistulas follow any definite pattern of arrangement with reference to the anal canal?

Some years ago Goodsall observed "that fistulae with an external opening situated behind an imaginary line passing transversely through the center of the anal opening, usually have the internal aperture in the midline and posteriorly, so the track is curved. On the contrary, where the external opening is anterior to the transverse line, the internal one, as a rule is immediately opposite, hence the track is straight."[17] This observation has come to be known as Goodsall's rule, and is represented diagrammatically in part 3 of Figure 2 which has been modified from Gabriel.[9]

As a possible means of explanation of Goodsall's rule let us examine briefly the lymphatic drainage of the anal, perianal and perineal tissues.

Figure 2, part 2, is modified from the author's thesis.[14] The lymphatics of the anal and perianal tissues in the newborn were filled with mercury using the "multiple puncture" method. It can be seen that all the collecting vessels, whether they arise posteriorly, laterally or anteriorly from the anal network of origin, eventually pass anterolaterally on their way to the regional inguinal nodes. This work confirms the observations of the classical authors in this field. Since the lymphatics play an important rôle in the spread of infections, it would seem logical to believe that an anal fistula arising in the dentate

margin posteriorly would tend to follow the course of the collecting lymphatic vessels which course, posteriorly and laterally, is curved. By the same reasoning a fistula arising in either of the two anterior quadrants would be likely to follow a fairly direct course to its secondary opening.

DIFFERENTIAL DIAGNOSIS

Under this heading let us consider the other, but less common, types of fistulas and sinuses which may involve the anal, perianal and perineal skin. Brief mention will be made of the essential features which might factilitate differentiation of these entities.

For didactic purposes let us consider these subjects first in outline form:

1. Anal fistula in association with anal fissure
2. Anal fistula of specific etiology:
   - (a) tuberculosis
   - (b) actinomycosis
   - (c) venereal lymphogranuloma
   - (d) malignancy
3. Rectovaginal fistula (anovaginal)
4. Anal fistula in acute dysenteries (e.g., chronic ulcerative colitis)
5. Anal fistula accompanying regional ileitis, etc. (Crohn)
6. True sinuses:
   - (a) true anal sinus, so-called incomplete fistula
   - (b) penetrating injuries—impalement, enema tips, etc.
   - (c) osteomyelitis of sacrum, coccyx or pelvic bones
   - (d) foreign body
   - (e) pyoderma, or hidradenitis suppurativa, etc.
7. Perineal sinuses:
   - (a) lymphatic abscess (Buie)
   - (b) urinary tract—periurethral abscess
   - (c) Bartholin cyst abscess

At the distal margin of a chronic posterior anal fissure one frequently finds a large skin tab known as a "sentinel" tag or pile. This tag is the result of inadequate drainage at the outer margin of the fissure. As infectious material continues to be caught beneath the outer margin of the fissure, a tiny abscess may form beneath the skin. Spontaneous rupture provides a secondary opening immediately distal to the sentinel tag. Thus a true fistula is established—a short subcutaneous tract arising at the distal margin of the fissure and ending 1 or 2 cm. further distally. It is not, however, an anal fistula proper since it does not arise in the dentate margin. This lesion can be identified easily at the time of surgery.

In tuberculous anal fistula the Mycobacterium tuberculosis is usually a secondary invader. Buie, N. D. Smith and Jackman[7] believe that the diagnosis is best made by means of properly done inoculations of guinea pigs. On the other hand C. L. Martin and Sweany[13] state that positive guinea pig tests are of value only in patients who present negative sputa. These authors advocate histopathologic study as the only pathognomonic test.

Involvement of anorectal tissues by actinomycosis is rare. However, one should be able to find the typical "sulphur-granules" in the purulent material from an actinomycotic lesion, and to demonstrate the ray fungus on microscopic examination.

Among the protean manifestations of venereal lymphogranuloma are anal fistulas, especially in the female in whom the genito-anorectal syndrome is more likely to be found. Attention has already been called to the probable rôle played by the lymphatics in the development of an anal fistula. Since the causative virus in venereal lymphogranuloma is usually spread via the lymphatics, one might expect anal fistulas to occur, especially where an inflammatory rectal stricture exists. Infectious material from the ulcerated and narrowed rectum could easily enter the perianal tissues through the anal crypts and anal ducts. Rectovaginal fistula occurs frequently in these patients together with elephantiasis (lymphedema) of the external genitalia. The specific intracutaneous test of Frei should be done using several different antigens if necessary.

Rosser[18] has called attention to the development of a malignant process in a

fistulous tract and believes that the fistula may act as an etiologic agent. N. D. Smith,[20] however, suggests that the fistula develops secondarily, and this has been the author's experience. Thorough examination, under regional anesthesia if necessary, together with microscopic study of tissue specimens should help to clarify the picture.

True rectovaginal fistula is a communicating pathway between the rectum and the vagina through any part of the rectovaginal septum, and is not within the scope of this paper. The more common form of rectovaginal fistula is, in reality, an "anovaginal" fistula inasmuch as the primary opening lies in an anterior anal crypt. The secondary opening is usually found in or near the posterior margin of the vaginal introitus. The identification of the lesion should not be difficult for the physician who fully understands the mechanism of development of an anal fistula. Careful bidigital palpation of the tissues is important. Probing, as stated previously, is more easily accomplished when regional anesthesia has been established.

Anal abscess and anal fistula are by no means uncommon complications in diarrheal disease such as chronic ulcerative colitis.[24] This is readily understandable when one considers the ease with which the liquid and highly infectious discharges can gain entrance to the anal crypts and anal ducts. The diagnostic problem is no different here than in the ordinary type of anal fistula. It is extremely important, however, that a complete proctoscopic examination be done in order that the presence of colonic disease be discovered. Patients harboring chronic ulcerative colitis do not tolerate surgery well, and anorectal wounds heal poorly if at all. The doctor is justified in establishing drainage for an anal abscess, but he should refrain from any deliberate anorectal surgery while there is the slightest evidence of active disease in the rectum and colon.

Penner and Crohn[15] in their study of regional ileitis have demonstrated fistulous processes which arise in the ileal area and terminate in the perianal or in the perineal skin. The demonstration depends upon roentgenologic visualization of the tract after it has been filled with lipiodol. The authors admit that the proof of such a process is difficult, and state that anal fistula accompanying regional ileitis may be the ordinary type.

A true anal sinus as shown in Figure 1, part 4, can be said to exist when an anal abscess has formed but has not ruptured spontaneously or has not been incised. It is possible for the abscess to become partially drained through its primary opening with some relief to the patient. If sufficient drainage occurs in this manner, such a sinus becomes a chronic process subject, however, to acute flare-ups should the primary opening become occluded. Since the opening of the sinus lies in the dentate margin, it can be seen only on internal (endoscopic) inspection at which time a drop or two of pus may be seen oozing from the involved crypt. Careful palpation should afford the examiner valuable evidence. At the time of surgery a chronic anal sinus can easily be rendered a complete fistula and then treated accordingly.

In the instance of a penetrating injury the history is usually significant. When the penetrating object enters the perineal or perianal tissues, a draining sinus may result the extent of which can be determined only by the use of lipiodol and subsequent roentgenologic visualization.

In most enema tip injuries the damage involves the skin of the perineum, the anterior anal wall or the anterior wall of the lowest one-third of the rectum. We are concerned here with the injury resulting from false passage of a hard enema tip beneath the perineal skin. There results from this type of trauma a sinus beginning in or near the anterior anal margin and extending anteriorly beneath the skin of the perineum. With the establishment of proper anesthesia such a lesion can be readily explored by means of a blunt probe. The tract should be widely uncovered.

Bacon and Taylor[1] report two cases of osteomyelitis involving the sacrum and

coccyx and accompanied by sinus formation simulating anorectal fistula. These authors stress the importance of *x*-ray in the diagnosis. According to N. D. Smith:[21] "Osteomyelitis of the bones of the pelvis, of pyogenic or tuberculous origin, may also cause the formation of an abscess and a subsequent persistent sinus or sinuses in this region. Grossly, such sinuses may resemble closely a fistula-in-ano. Lack of a demonstrable primary opening at the dentate margin, a palpable extra-rectal inflammatory mass attached to the bony pelvis and roentgenographic demonstration of alteration of the normal bony structure of the involved portion of the pelvis will provide the diagnosis."

Numerous objects, such as chips of bone, surgical needles, etc., have been reported as etiologic factors in persistent perineal and perianal sinuses. The writer has seen a rubber drainage tube with an attached safety pin removed from the ischioanal fossa during the surgical exploration of a longstanding draining sinus. Usually there is a history of previous attempts at anorectal surgery.

Since apocrine sweat glands are found in the genital and perianal areas as well as in the axillary, mammary and inguinal regions one must consider hidradenitis suppurativa. This entity, according to Brunsting,[3] "is characterized by the formation of abscesses and sinuses in selected regions of the cutaneous surface in which the apocrine type of sweat glands are situated. The favorite sites of involvement are the axillary, mammary, inguinal, genital and perianal regions. The disease affects young adults, usually occurring in the second decade of life. The course is extremely chronic and frequent remissions and relapses occur. The essential histologic features include primary involvement of the apocrine sweat glands and dissemination of the disease throughout the subcutaneous tissue by means of the lymph channels."

In a consideration of the causes of perineal sinuses in the male one must not overlook disease and deformity of the urinary tract. In rare instances a persistent perineal sinus may result from a prostatic abscess or from perineal prostatectomy.[8] Urethral stricture may become so marked as to cause urinary extravasation necessitating incision for drainage. Sinuses will persist until the urinary tract obstruction is relieved. Urethral instrumentation may result in periurethral abscess and subsequent fistulous communication between the urethra and the perineal skin. Abscess of Cowper's gland may be the underlying cause of a perineal sinus. Hypospadias must be considered as a possible explanation of a urinary fistula opening upon the perineum. A careful history and a thorough urologic investigation will serve to establish a proper diagnosis.

Buie[4] has called attention to draining sinuses involving the perineum, usually in males, which simulate anal fistulas. There is usually a history of multiple operations, and it is difficult to identify any one anal crypt as the probable source of the process. Buie has obtained satisfactory results in these patients by excision of all the anterior anal crypts, the excision including all the skin between the anus and the secondary openings in the perineum. He believes, therefore, that this process is the result of anal infection with invasion of the superficial perineal tissues by way of the lymphatics which drain the anterior wall of the anal canal. Tucker, in discussing Buie's paper,[4] called attention to the probable rôle of the anal ducts in anal infection.

Although a Bartholin cyst abscess may be mistaken for an anal abscess, it seldom leaves a persistent draining sinus following spontaneous rupture. A persistent sinus, therefore, is evidence in favor of a true anal fistula. Careful inspection and palpation of the tissues together with anoscopic examination usually afford the proper diagnosis.

SACROCOCCYGEAL SINUSES

Although several interesting theories have been brought forth in explanation of sinuses, cysts and tumors of the sacrococcygeal region most authors agree that the complex developmental anatomy of the region favors these abnormalities. Stone[25]

thinks that pilonidal cyst may be the homologue of the "preen gland" in birds.

Pilonidal cyst is the most common of the lesions in the sacrococcygeal region which are developmental in origin. The adjective "pilonidal" is derived from the Latin words "pilus" meaning hair and "nidus" meaning nest. The cyst communicates with the surface of the overlying skin by means of one or more sinuses (Fig. 2, parts 4 and 5) which lie usually in the midline anywhere along the line AB. Although openings may be found to either side of the midline, and lateral to or even anterior to the anus, these additional openings are usually the result of infection of the cyst cavity. The author believes that the term "pilonidal sinus" should pertain only to the sinus openings and tracts, and not to the cyst itself, but common usage would indicate that the terms "pilonidal cyst" and "pilonidal sinus" are considered to be synonymous.

The usual patient harboring a pilonidal cyst is a young man of the "hairy" type in his late teens or early twenties who complains of a painful swelling at the base of his spine. There may or may not be a history of injury. The patient may be suffering from the "abscess" for the first time, or he may report that he has had several abscesses in this region, each one accompanied by painful swelling and followed by relief after spontaneous rupture or after incision for drainage.

Upon examination the physician finds the usual signs of an acute abscess involving the skin and subcutaneous tissues in the sacrococcygeal region. If he looks carefully, he may discover one or more small, round openings or "dimples" in the skin in the midline. There may be tufts of hairs protruding from these openings which can be easily withdrawn with forceps. The hair is usually quite long and is "silky" in appearance. The finding of such hairs is almost pathognomonic of a pilonidal cyst in which secondary infection has developed. Scars may be found indicating previous incision for drainage. The abscess should be widely incised.

Pilonidal dimples may be found accidentally during the course of a routine examination. The examiner should be particularly alert when performing his external inspection of a patient of the hairy type. Knowledge of the presence of a pilonidal cyst is of value to the surgeon who uses sacral anesthesia in his anorectal surgery. The author feels justified in assuming that every pilonidal cyst is potentially infected and that, therefore, sacral anesthesia for some anorectal procedure is contraindicated.

It is believed that here again, as in the instance of sinuses or fistulas elsewhere, the use of probes should be postponed to the time of surgery when suitable anesthesia has been established. Careful probing enables the surgeon to outline the extent of the cyst, and he should attempt to carry his excision well into normal tissue. Dyes are unnecessary. The excised tissue is examined again with probes and should a probe easily pass through the base of the tissue, indicating a perforation of the wall of the cyst, the corresponding area in the base of the wound can be explored for gross evidence of "pilonidal" tissue. The pathologist usually does not like to have his specimen incised by the surgeon at the operating table, but there is no better method of teaching the surgeon the gross characteristics of "pilonidal" tissue, i.e., the greyish color and the gelatinous nature of the material within the cyst. These features together with the presence of hair in the cyst usually leave no doubt as to the diagnosis.

DIFFERENTIAL DIAGNOSIS

The secondary openings of anal fistulas may occur posteriorly in the sacrococcygeal region, and pilonidal sinuses may be found lateral to, and even anterior to, the anus. The diagnostic features of both entities have been indicated above.

Disease of the underlying sacrum and coccyx must be considered, and has already been discussed.

Needless to say the presence of any sizable presacral lesion should be disclosed by a careful digital examination of the

rectum. A retrorectal inflammatory process, anal in origin, must be considered.

Sinuses in the sacrococcygeal region due to chromaffin bodies, and sinuses due to dermoid cysts anterior to the sacrum are rare according to N. D. Smith.[22] Very little inflammatory change is found in the sinuses accompanying a chromaffin body, and the wall of the cyst (upon excision) is bright yellow in color. With regard to dermoid cysts Smith refers to roentgenologic study and to the discharge of sebaceous material from the sinus.

Another type of cyst lying anterior to the sacrum is the so-called "multilocular" cyst[23] with a sinus lying immediately posterior to the anus. The content of the cyst is a clear, gelatinous fluid and there is usually a history of several operations.

Thus far in his experience the author has not seen a presacral cyst or tumor.

Thomason[26] has divided the tumors and cysts of the sacrococcygeal region into those which lie dorsal to the sacrum and coccyx and those which are situated ventral to these bony structures. According to this author the pathological change in pilonidal cyst is simple, whereas ventral cysts and tumors are prone to undergo malignant change with spread by extension and invasion rather than by metastasis.

SUMMARY

In accordance with the accepted definitions of "sinus" and "fistula" an attempt has been made to indicate those lesions which are true fistulas and those which are true sinuses in the anal, perianal, perineal and sacrococcygeal regions.

Inasmuch as the great majority of so-called "draining sinuses" in the anal, perianal and perineal skin are in reality the secondary openings of true anal fistulas, an effort has been made to clarify the pathogenesis of anal fistula. It is hoped that a proper understanding of this mechanism will lead to more accurate diagnosis and to adequate management.

Pilonidal cyst is the most common "developmental" lesion dorsal to the sacrum and coccyx.

The essential differential diagnostic features of anal, perianal, perineal and sacrococcygeal sinuses have been briefly stated.

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LYMPHOGRANULOMA VENEREUM*

WITH SPECIAL REFERENCE TO RECTAL STRICTURE

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DURING the past decade and a half the pages of medical literature both here and abroad have been strewn with references to lymphogranuloma inguinale. The disease is of interest by virtue of the fact that we realize it is not new; that many cases of inguinal buboes have been and are manifestations of this disease; that inflammatory rectal stricture, which is encountered more commonly in women, is most frequently the result of a perirectal lymphangitis initiated by the specific virus of lymphopathia venerea; and that esthiomene, chronic elephantiasis or the combination of these, the genito-anorectal syndrome,[1] or the anorectal syphiloma,[2,3] are part of the same infection.[4–8]

Durand, Nicolas and Favre,[9] in 1913, offered the first detailed account of this disease although Heiner[10] reported eighteen cases in the same year. Referring to the pathologic process as "subacute lymphogranuloma inguinalis" they presented clinical and histologic data in an effort to prove that this disease was an independent entity. They held that it was contagious and of an infectious nature, which view is also stressed by Nealton.[11] In their description they mention that the disease is of genital origin, that the initial lesion is frequently unobserved but that the inguinal adenitis is a constant and prominent feature. Even though these workers did not appreciate the close analogy between this and climatic bubo, which since has been shown to be identical,[12,13] they did realize that this pathologic process was not a new disease.

Lymphogranuloma venereum or lymphopathia venerea is an infectious disease usually of venereal origin caused by an unknown filtrable virus, characterized in the male by a somewhat insignificant initial lesion, which is followed by a suppurative inguinal adenitis, elephantiasis of the penis and scrotum and, less frequently, stricture of the rectum; in the female by stricture of the rectum, abscess, fistulas, anovulvar esthiomene and elephantiasis.

Many terms have been applied to this malady. The tortuous course this disease has taken in tracing its evolution through the history of medicine is indicated to some degree in the unusual names that have been applied to it in this country or another. They imply a lack of agreement among scientists owing perhaps mainly to the fact that they have been working more or less independently as to the nature, etiology, manifestations and significance of this condition. Those in general use are "lymphopathia inguinale" so called by Hellerstrom[14] and "lymphopathia venera" as suggested by Wolf and Sulzberger.[15] Other names employed are lymphogranulomatosis inguinalis,[16,17] lymphogranulomatosis venereum,[18] subacute lymphogranuloma inguinalis,[9] simple subacute adenitis with purulent intraganglion, nontuberculous granulomatous lymphadenitis,[19] inguinal lymphadenitis,[20] hypertrophic bubo,[21] tropical bubo, strumous bubo[22–25] climatic bubo,[26,27] malarial bubo, pestis ambulans, poradenolymphitis, poradenitis,[28] Nicolas-Favre's disease,[29] fourth venereal disease,[30,31] and sixth venereal disease.[32]

It was believed at first that the disease confined itself, or predominated in, the male; but observations, plus the obscurity

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of the primary lesion in the female as well as the supression or deviation of pathologic manifestations, argue that lymphogranuloma venereum is, comparatively speaking, equally distributed between the sexes. To a great extent, the ratio is dependent upon the source of the series; for instance in our rectal clinics the preponderance is in the female although our colleagues in the genitourinary clinics observe a greater incidence in the male. Our series as reported in early 1941[33] was as follows: females, 567; males 285; total, 852.

The age incidence is usually from fifteen to forty-five or the period of greatest sexual activity. Isolated cases have been reported in children,[52,107] but in no such case was sexual irregularity or abnormality observed. In our series of cases, the greatest incidence occurred between seventeen and forty; three cases under the age of one were noted, the youngest of which was three weeks.[34,35]

INCIDENCE ACCORDING TO AGE

Below 1 year	9
14–29	301
30–39	368
40–49	98
50–59	51
60–69	17
70–79	5
80–89	3
Total	852

Early reports seemed to confine this affection to tropical races, notably negroes, but with the awakening of interest and observation in nordic countries some have come to believe that race plays no significant part in the incidence.[36] As shown by the accompanying table, the incidence in the colored race was much greater in the author's series and this seems to be the usual experience of investigators in this country.[112,113,114]

Colored	559
White	292
Chinese	1
	852

Occupation. Early observations practically confined this disease to sailors and the lower classes inhabiting port cities; it was, therefore, believed to be carried from tropical regions on board vessels. Since, however, the study of this affection has been undertaken more or less universally, reports mention patients from nearly all walks of life. Gray and Yieh,[37] in a report from China, list students, housewives, hawkers, merchants, actors, soldiers, sailors, coolies, printers, blacksmiths, etc., with the highest number equally divided between coolies and housewives. In France, Flandin and Turiaf,[38] in a series of four, treated two clerks, a mechanic and a machinist. Hellerstrom noticed a predominance in ports; Stannus believes that the apparent high frequency in sailors is not a true occupational incidence while the lack of special comment on this phase by American observers would argue that the occupational incidence is so distributed as to justify no dogmatic conclusions as to any one class.

The mode of transmission is primarily and principally coitus. In this, experience justifies the inclusion of buccal coitus, and the practice of suction penis and cunnilingus. Scattered cases, moreover, evidence the possibility of extragenital infection as well as extragenital lesions.[40–51,53,54,60,107–111,115] In the rare cases of the children,[52] it was found that the mother had been infected and had used her wash cloth, enema tip, or other personal accoutrements on the child as well as having the child sleep in the same bed with her. Stannus[55] mentions two sisters, aged six and seven, respectively, who contracted the disease, manifested by inguinal masses the size of hens' eggs in one and ducks' eggs in the other, through the simple, ordinary contacts of living in the same house with an infected person. Physicians have become infected by surgical contact with these patients;[22,56–58] an instance has been cited in which the infection appeared first on the finger and later in the axillary glands.

The existence of two types of virus or of a double-acting nature in the single virus of lymphopathia venereum is a general

theory among those who are making a close study of this disease.[59] Although not yet identified, the virus is capable of passing a Berkefeld V and a Chamberland L3 candle. It will not pass an ultrafilter.[6] It has been found to survive at 2° to 3°C. below zero for ten days; at 4°C. for twenty-three days; at 10° to 20°C. for thirty days; at room temperature for twenty-four to forty-eight hours; at 46°C. for up to thirty minutes; at 56°C. for ten minutes; it is destroyed at 60°C. It loses its virulence in 0.1 per cent formol, in glycerin, and upon prolonged desiccation. Miyagawa and his associates[62] found it still active after thirty day's but inactive after thirty-five days' desiccation. Tamura[63] reports cultures made on Tyrode's medium, one being carried through twenty-four subcultures.

Lymphopathia venereum is freely and reciprocally inoculable among men and animals. The best experimental animals are mice, preferably white, guinea pigs, and monkeys, preferably the hepale penicillata or marmoset. Phylactos[64] has successfully inoculated rabbits in the cornea and believes that these animals promise to be equal or perhaps greater in value for experimental and diagnostic purposes than monkeys, which are costly and hard to obtain. Some strains become more virulent at each passage. Chatellier[65] has accomplished at least twelve passages of one virus; the Kamm strain, the most virulent, has been passed through twenty monkeys without attenuation. Conversely, the intradermal injection of a single strain into one patient becomes less potent at each injection. Microscopic study of inoculable material yields no distinctive characteristics. Rodlike bacteria, not unlike the Donovan bodies of granuloma inguinale, have been observed.[48,66,67]

Antigen prepared from animals is equally as valuable as human antigen. Lichtenstein and Von Haam[68] state that "brain emulsions of white mice and of monkeys (Hapale penicillata) infected with the virus of lymphopathia venereum provide a large supply of a uniformly potent antigen for the diagnostic intracutaneous test. There is no danger of contamination with pathogenic organisms, particularly of the spore-bearing type, as there may be when pus from spontaneous buboes in humans is employed directly for the preparation of the antigen. Finally, the false positive reactions obtained occasionally with the Frei test are completely eliminated by this method." Nonfiltrated antigen is the more virulent. It is stated as a general fact that after two months the antigen is not reliable for diagnostic purposes, although scattered instances of reactions obtained with antigen older than this are recorded, Hellerstrom having used successfully an antigen over a year old.[69] It has been our experience that the Frei antigen is most active until about the third month and then diminishes in its potency until the ninth month after which it is usually inactive.

Inflammatory Stricture of the Rectum. Stricture of the rectum is an organic narrowing of the lumen of the bowel by fibrous tissue involving the mucous membrane, submucosa and muscular coat and characterized by progressive constipation, tenesmus and mucopurulent discharge. In 1931, the writer[70] noted the site in a series of 179 cases as follows:

	No. of Cases		Per Cent
Sigmoid	6	or	3.4
Within 5th inch above anorectal line	8	or	4.5
Within 4th inch above anorectal line	5	or	2.7
Within 3rd inch above anorectal line	23	or	12.8
Within 2nd inch above anorectal line	65	or	36.3
Within 1st inch above anorectal line	49	or	27.4
Anal—at or below anorectal line (stenosis)	23	or	12.9
	179		100.0

The incidence as to age, sex and race is as follows:

Age	No. of Cases
10–20	31
21–30	194
31–40	230
41–50	157
51–60	14
61–70	9
Over 70	4
Total	648

Sex	No. of Cases
Females	537 or 82.8%
Males	111
Total	648

Race	No. of Cases
Colored	447 or 68.9%
White	201
Total	648

Strictures of the rectum are classified according to their shape, as annular or tubular. The annular variety represents a ring-like constriction involving the entire circumference of the rectum; the tubular variety is so specified to denote a cannular or tube-like contraction of the entire circumference of the rectum.

Pathology. By way of the extensive lymphatic network and from initial lesions of the fourchette and vagina, invasion with the specific virus results in a nutritional interference in the regions draining into the adjacent glands. Lymphostasis is probably the result of the virus of lymphogranuloma venereum. It should be borne in mind that the inflammatory process may attack any layer of the rectum or the tissues outside its wall. If it is initiated from within, the irritation results in erosion of the mucous membrane upon which infection is superimposed. With continuation of the etiologic irritant the inflammatory process becomes subacute and finally chronic in nature, so that the various layers of the rectum and the tissues outside its wall are gradually involved by continuity and contiguity of structure. As a result of this chronic inflammation much young fibroblastic tissue is deposited in the submucosa as well as in the other coats, which gradually leads to thickening of the visceral wall. This in itself tends to encroach on the lumen of the rectum. By subsequent contraction of the maturing fibroblastic tissue, this thickening becomes markedly increased so that there eventually results a firm, inelastic, permanent narrowing to which the term stricture is applied.

On the other hand, if the initial focus is outside the rectum, as we believe is most frequently the case, the extramural network of lymphatics becomes invaded. As the inflammatory process gradually becomes chronic the mural tributaries, namely, the inter- and intramural groups, are invaded by extension. As a result of the inflammatory process, fibrous tissue is deposited in the various layers of the rectum so that thickening occurs which brings about narrowing of its lumen. As the process continues and additional fibroblastic tissue is deposited, subsequent contraction ensues so that finally an organic stricture is formed.

At first, erosions of the mucous membrane are noted, followed by ulcerations, so that the surrounding mucosa appears altered and somewhat lusterless. To the touch, the involved area is thick and firm, while later it feels leathery, with loss of elasticity and distensibility. It is more or less irregular, markedly thickened, and the mucous membrane is found to be adherent to the tissue beneath. In the deeper layers of the stricture, fibrous tissue is seen involving all the coats of the rectum, although the greatest amount of involvement is in the submucosa. Fistulous tracts may be found passing to the perirectal tissues, to adjacent structures as the bladder, urethra, vagina or through the skin. Ulceration is usually marked and occurs early. The discharge is frequently abundant, mucopurulent and often sanious. Ulceration rarely exists at the level of the stricture except in the tubular variety, where we see it routinely through the stricturoscope and at autopsy. Above the stricture, the rectum is dilated and ballooned owing to the pressure of feces. In this location, ulceration is frequent because of irritation from the retained fecal contents and bacterial activity. Polypoid growths are not uncommon in this site. Below the stricture, the mucous membrane appears gray, it is tough and dense with loss of the normal velvety character. Here ulceration is less common. The mucosa and anal skin are often thrown into patchy thickenings and papillomatous vegetations

which may project into the lumen and through the anal orifice.

The histopathologic appearances of inflammatory rectal stricture are nearly always nonspecific regardless of etiology, save in the occasional case in which typical tuberculous granulation tissue is characteristic. In lymphogranulomatous strictures one finds small abscesses which are observed uniformly in the lymph-node. Ordinarily the following changes are noted: the ulcerated mucosa is replaced by simple granulation tissue; the entire wall of the rectum is more or less heavily infiltrated by a chronic inflammatory cellular exudate in which lymphocytes and plasmocytes predominate, monocytes and macrophages being in the background; a zone of neutrophilic leukocytes lies along the lumen margin and may, with eosinophils, be sprinkled sparsely throughout; fibroblastic proliferation is prominent in all coats of the bowel, later forming a dense, collagenous mat which may largely replace the original structures; blood vessels show the proliferative thickening common to any chronic inflammatory area and the environs of the lesion are usually surrounded by a round-cell collar.

Jones and Rome[116] investigated a series of seventy-three patients with lymphogranuloma venerea on whom a total of ninety-three protein determinations were made. Sixty-four per cent had total proteins greater than 8 Gm. per cent but more characteristically approximately 90 per cent showed globulin values greater than 3 Gm. per cent. In 32.5 per cent an albumin of less than 4 Gm. per cent was noted. Fifty-two per cent showed a reversal of the albuminis-globulin ratio and this reversal was always due to an increase in the globulin fraction with or without a decrease in the albumin fraction. In the sixty-five determinations, whose values lay in the normal range between 6–9 Gm. per cent total protein, fifty-five showed an increase in the globulin fraction. This increase in globulin was due to an increase in the euglobulin portion of the globulin fraction.

Abnormally high globulin values have been noted in a few cases, in whom the globulin level fell to normal figures within one or two weeks and in one case there was a subsequent rise to a level higher than its previously noted hyperglobulinemic state.

The Takata-Ara reaction was positive in half of the cases without demonstrable evidence of a pathological condition in the liver. This reaction is likely to be positive in instances of hyperglobulinemia and offers a simple method for the detection of this alteration.

Symptoms. In all cases the symptoms vary with the degree of completeness of the constriction, and this is usually dependent upon its location and duration. Initially, a bearing-down sensation or a feeling of discomfort in the rectum frequently exists. Constipation is progressive and a constant desire for stool is cited by the patient so that futile and painful straining (tenesmus) soon ensues. The evacuations, which are always incomplete and which are increased by the use of various drastic purgatives, become liquid and tinged with blood so that, as the condition progresses there is an almost constant dribbling of mucus, pus and feces. Many writers stress the occurrence of ribbon-shaped stools in stricture of the rectum, but in our experience this is uncommon except in those instances in which the degree of constriction is marked and in which it is located immediately above the anorectal line. In anal stenosis, however, ribbon-shaped stools are of frequent occurrence. Soreness about the anus is frequently present owing to the excoriation caused by the irritating discharge. Concomitant gastrointestinal symptoms, as anorexia, meteorism, coated tongue, as well as loss of weight and general impairment in the health of the individual, are later manifestations. Not unimportant is the previous history, especially of anorectal and pelvic operations or disorders. In a series of 216 cases of which 173 were females, 143 or 82.6 per cent gave a history of previous pelvic inflammatory disease. A history of syphilis, either the initial

lesion or constitutional lues, was given in forty-two cases.[71]

Diagnosis. A history of constant soiling by feces, blood and pus is suggestive of an inflammatory stricture, especially when cited by a colored female between the ages of twenty and forty years.

Inspection. Although the diagnosis by inspection is not absolute, to the careful observer it offers more than a suspicion of the pathological condition present. Not infrequently the region about the anus is moist and glued together by the thick mucopurulent discharge. Upon separation of the buttocks, fecal matter mixed with blood and pus may be seen seeping through the anal orifice. Hypertrophied skin tags, condylomas of various sizes and one or more fistulous openings are not uncommon.

Digital Examination. Since approximately all inflammatory strictures of the rectum are within reach of the finger, the diagnosis is not difficult. As the gloved finger is inserted into the anal canal some degree of muscular relaxation is noted in longstanding cases, owing to fatigue of the external sphincter. As the finger is advanced the stricture is felt as a firm, inelastic narrowing, usually involving the entire circumference of the rectum. Through an ordinary proctoscope, the stricture (or in the case of the tubular variety, the lower border of the stricture) is noted by its pale, leathery and thickened appearance.[72] The introduction of the stricturoscope should be cautiously performed, preferably with the patient in the inverted position and with the instrument being advanced under direct vision. In this manner the type of stricture, whether annular or tubular, its length and its degree of ulceration may be determined.

In each case of stricture, especially when located high in the rectum or sigmoid, roentgenograms should be taken after an opaque enema. In this fashion, the irregular deformity will be noted. The method devised by Martin, Sturr and Bacon[73,74] has proved of distinct value in estimating the accurate length and site of rectal strictures. A soft rubber catheter of small size, calibrated in centimeters by strips of lead foil, is encased in a rubber balloon and inserted into and through the stricture. The proximal end of the balloon is tied so that no barium will escape after its introduction through the catheter. Under the fluoroscope, the barium or other opaque solution is permitted to run in until the stricture is clearly outlined. Films are then taken and the length of the stricture as well as its distance from the anal margin can be readily calculated by means of the lead strips.

The Frei Test. One-tenth cc. of the antigen is injected intradermally into the arm of the patient. The reaction, which reaches its maximum from forty-eight to seventy-two hours after the injection, is then read. A positive test is characterized by the appearance of a hard red papule around which may be noted an erythematous ring. In some cases and especially when stronger dilutions are used (1 to 5), pustule formations occur as shown by a central area of necrosis.

Control. In order to evaluate the test properly, a control antigen obtained from normal glands of patients not affected with lymphopathia venerea should be employed. A sterile solution of leukocytes has been used also. Wang[75] suggests a control of phenol, 0.25 per cent, in normal saline solution. The material is diluted with physiologic salt solution as in the preparation of the specific antigen, after which 0.1 cc. is injected into the forearm of the patient. It has been our custom to inject the Frei antigen in the left forearm and the control in the right in order to avoid incorrect readings. When the control test is noted, which is at the time the Frei antigen is read, or forty-eight to seventy-two hours after injection, no elevation, pustule or erythematous zone should be present. Such constitutes a negative control test.

Male patients with this disease who present inguinal buboes will show a positive Frei reaction if tested one week

after onset of the adenitis, although the usual time is two weeks to two months. Cole[76] observed a positive test four days after appearance of the bubo and eighteen days after intercourse. In our series the earliest was twenty-one days after exposure.[77] Hellerstrom reports a case in which a positive reaction appeared four months after onset of the adenitis, having been negative during the second and third months. This investigator was able to prove that cutaneous manifestations were positive from eleven to twenty-four years after the appearance of the adenitis, and Cole observed a similar case at thirty years. In all probability the reaction, having once been proved positive, will remain so for life.

Antigens prepared from experimental animals (monkeys, guinea pigs, and mice) are equally as specific as the human antigen for diagnostic purposes and even for treatment. This fact, together with the ability to preserve antigen in concentrated form in dried pus, assures a sufficient and reliable quantity. In regard to preservation Grace[90] declares, "The titration of Frei antigens prepared from fresh and dried pus, respectively, has been carried out in eight normal and eleven lymphogranulomatous persons. The results obtained show a slight difference in titer in favor of the dried material."

The diagnostic value of the Frei test is now generally recognized, but its specificity, though practically 100 per cent, is still subject to doubt in some few cases. The author[77] in a previous article, tabulated the percentages of positive reactions as observed by various investigators, the majority showing 100 per cent and most of the remainder above 90 per cent. At the opposite extreme, a very recent article by Charles Flandin and Jude Turiaf, of the Hôpital Saint-Louis, Frances, states, "The opinion which we have reached is in accord with the conclusions announced by Ravaut in the thesis of his pupil, Maisler. The action of the lymphogranulomatous antigen is very inconstant."

Aside from microscopy of the tissue, the Frei test is, nevertheless, the most dependable factor in the diagnosis and the antigen is also valuable for treatment. It should be remembered that a negative result does not necessarily predicate freedom from contamination. The reaction may be negative in the initial phases of the disease, appearing later on when the infection has progressed from the lymph glands to the skin. In accompanying tuberculosis and syphilis the Frei reaction may be suppressed either partially or totally.

Source of Material for Performing the Frei Test. The material for the Frei antigen is obtained from patients with lymphopathia venereum as proved by a positive Frei test using a known positive antigen.[77] To prepare the antigen, either the pus or tissue may be used. Buboes that are unopened and present areas of fluctuation are preferable for obtaining purulent material. We have used successfully tissue from the anovulvar region of patients showing an esthiomenic process, fragments of rectal stricture, and pus from perirectal abscesses and fistulas occurring in cases with a positive Frei test.

Preparation of Frei Antigen. After the addition of physiologic salt solution as a diluent, the material collected is heated on a water bath at 60°C. for two hours one day, and at 60°C. for one hour the second day. Tests for sterility are performed on aerobic and anaerobic culture media and, if found sterile, the material is sealed in a sterile ampule ready for use. The antigen is then tested on known positive cases of lymphopathia venereum as previously determined by the obtaining of Frei reactions.

Ottilino[104] has devised an intracutaneous test using the cerebrospinal fluid of Frei-negative men. The fluid and the vacuum concentration is performed under aseptic conditions. By means of a graduated pipette, closed at one end and submerged at the other in the fluid contained in a porcelain capsule, the degree of concentration is determined. By reducing 10 cc. of the fluid to 1.5 cc., it resembles a col-

loidal solution, is opalescent and foams on agitation. Otilino found that if this concentrated fluid is injected intradermally, a vesicle appears in twenty-four hours which he terms "vesicular test." The shape is described as ovoid, circular or triangular with rounded angles but there is definite trend toward the hemispheric form. It measures 4 mm. in length, 2 mm. in width and 1 mm. in height. The color is light or dark brown, sometimes dark gray. This investigator is of the opinion that the concentrated cerebrospinal fluid in Frei-positive patients, when injected intradermally, has a certain antigenic power that provokes a blister reaction. The study of the vesicular test in different groups of patients showed that the test was positive in patients with poradenic virus infection, previously diagnosed by Frei's test.

INCIDENCE OF POSITIVE FREI TEST IN INFLAMMATORY RECTAL STRICTURE

Author	City	No. Inflammatory Rectal Strictures	Positive Frei Test—Per Cent
Curth[78]	New York	50	100
M. Hill[79]	Los Angeles	34	100
Dalton and Ricketts[80]	Indianapolis	15	100
Alley[81]	Lexington	20	100
Vander Veer[82]	Philadelphia	21	100
Grace[83]	New York	20	100
Cole, DeWolf, Van Cleve[76]	Cleveland	13	100
Lehman and Pipkin[84]	San Antonio	3	100
Streicher[85]	Chicago	11	100
Marino[86]	Brooklyn	4	100
Grossman[87]	Harrisburg	4	100
Hayden[88]	Boston	20	100
Howard and Strauss[89]	New Haven	5	100
Bloom[43]	New York	7	100
Coutts[59]	Chile	2	100
Wang and Shen[75]	Nanking	2	100
Templeton and Smith[91]	San Francisco	1	100
Rainey and Cole[92]	St. Louis	14	100
Lichtenstein[93]	New York	56	98
Martin and Bacon[94]	Philadelphia	155	96.7
Sulzberger[95]	New York	6	96.3
Rajam[96]	Madras	22	90.9
Bensaude and Lambling[97]	Paris	143	88
Lee and Staley[98]	Cincinnati	14	87
Stillman[99]	New York	6	83
Pennoyer[100]	New York	22	81
Corachan[101]	Madrid	3	75
Hayes and Burr[102]	Houston	106	60.4
Bacon (1939)[23]	Philadelphia	648	91.6

Differential Diagnosis. Ordinarily it is not difficult to diagnose a rectal stricture but to determine the cause or the process which bears influence is not always an easy task. The presence of amebiasis may be diagnosed by isolation of the Endamoeba histolytica; bacillary dysentery, by the characteristic bacterium and positive agglutination test of the blood; tuberculosis, by histologic and bacteriologic studies; syphilis, by means of the blood Wassermann, Kahn and Kolmer tests, and dark field illumination; gonorrhea, by isolation of the gonococcus of Neisser.

It should be mentioned, however, that in a large majority of stricture cases observed, a triad exists, viz., syphilis, gonorrhea and lymphopathia venereum. Tuberculosis, amebiasis and even malignancy may complicate the picture also.

DIFFERENTIAL DIAGNOSIS

	Nonmalignant Stricture	Malignant Stricture
Age	17–40	40–60
Race	colored	white
Sex	female	male
Palpation	firm but not nodular; orifice of stricture feels like a circular ridge	hard, nodular, edges thickened, irregular, induration is marked, new growth, more prone to cause obstruction
Involvement	entire circumference involved uniformly	more on one side than the other, not uniform
Odor	fetid	fetid and characteristic
Ulceration	above and below stricture	begins in growth
Metastasis	not present	present
Course and progress	chronic and slow	more rapid
Glandular involvement.	infrequent and less marked	always present, comparatively early
Loss of weight	gradual	pronounced and rapid, cachexia early
Laboratory		
Biopsy	suggestive of L.V.	positive for cancer
Gruskin test	negative	positive for cancer
Frei test	usually positive	negative

Not infrequently, the physician will be called upon to distinguish between an inflammatory and a malignant stricture. Usually a stricture caused by carcinomatous infiltration occurs in the white race between the ages of thirty-five and sixty. Here, the mass is hard and nodular and confined more to one side than the other; whereas the inflammatory stricture is firm and the entire circumference is involved

uniformly. In cancer, the mass grows rapidly and obstruction is frequent. Glandular involvement is noted and cachexia malignant type is usually fatal unless resection is performed early while the prognosis is good in lymphogranulomatous

DIFFERENTIAL DIAGNOSIS

	Inflammatory Stricture	Hyperplastic Tuberculosis	Sarcoma	Syphilitic Gumma
Age	17–40	20–30	30–50	middle life
Race	colored	white and colored	white	white or colored
Sex	females	males	males	females
Onset	insidious	insidious	rapid	insidious
Characteristics	uniform narrowing of entire, circumference, firm, inelastic, ulceration above, in and below stricture	pale and dry, thick, rigid; ulceration infrequent	hard or soft and spongy, begins as small movable nodule but later fixed	round or oval nodule—pea to an orange; single or multiple; painless; bluish or purple in color, firm to touch; movable and ulcerates regularly. Responds to antiluetic treatment
		tuberculosis elsewhere	cachexia and ascites, metastasis	manifestations elsewhere
Laboratory				
a. Intradermal test	Frei usually positive	tuberculin positive	Gruskin positive for sarcoma	
b. Stools	.....	tubercle bacilli only occasional		
c. Biopsy of tissue	not characteristic	miliary tubercles	positive for sarcoma	
d. Guinea pig inoculations	.....	shows tuberculosis		
e. Blood	.....	.....	.....	Wassermann, Kolmer positive

appears with loss of weight. Biopsy will confirm the diagnosis.

Complications and Sequelae. Usually inflammatory strictures are so progressive that, because of absorption of the retained products, severe toxicity ensues with loss of weight and general debility. Abscess and fistula, frequently multiple, are not uncommon and when present may rupture through the skin into the perirectal tissues, as the ischiorectal fossa and pelvi- or retrorectal spaces, or into adjacent structures, as the vagina, bladder and urethra. Peritonitis may occur from perforation of the ulcers above the peritoneal reflection or complete obstruction may take place.

Prognosis. In stricture of the rectum, the prognosis is variable and depends upon the etiology, extent, treatment instituted, age and condition of the patient. The strictures in which a radical two-stage operative maneuver is performed.

Treatment. It should be understood that in every case effort is made to ascertain the cause and any influencing factors; for instance, if the Wassermann reaction is positive, proper antiluetic treatment should be instituted, even though it has no effect on the fibrotic stricture. If the patient is tuberculous, measures should be directed to this angle. An attempt should be made to build up the constitution of the patient since there are usually varying degrees of weakness and loss of weight. As to the diet, it should be nutritious and composed of foods which leave but little residue. Vitamin therapy may be employed to advantage; ordinarily our patients receive B complex approximately 5,600 International Units daily.

Medicinal Treatment. Instillations of ichthyol, 10 to 20 cc. of a 25 per cent aqueous solution, twice daily, are soothing to the mucous membrane and will assist in diminishing tenesmus. Irrigations of hot water 110°F., potassium permanganate 1:8000, or S. T. 37 (hexylresorcinol) 1:8 once or twice daily, will tend to cleanse and heal the inflamed mucosa. For topical applications to the ulcerated areas, 5 or 10 per cent silver nitrate, metaphen or 50 per cent para-thio-cresol may be employed. Bleeding areas will often respond to a saturated solution of potassium permanganate. Easy passage of stools is facilitated by the administration of liquid petrolatum, ½ to 1 ounce, night and morning as indicated. In approximately 250 cases, sulfanilamide was given without noticeable results. Operative cases receiving this drug seemed to progress more satisfactorily. In almost every instance the period of convalescence seemed to be decreased. Eighty gr. were given by mouth daily for two days; thereafter, approximately 40 gr. daily for seven days. When ambulant, patients were given between 10 and 20 gr. daily but were frequently observed for untoward symptoms. Where this drug is employed, close co-operation with the medical department is to be advised. Recently, sulfathiazole has been used, and the results appear more satisfactory.

Surgical Procedures. Indications for operative intervention may be enumerated as follows: Marked constrictions not amenable to treatment, or those in which other measures have failed; long tubular strictures or those of the annular variety not within reach of the finger and above the peritoneal reflection, where obstruction is impending, and in the presence of marked constitutional symptoms.

Perineal Excision with Colostomy. No maneuver is permanent or ideal in the treatment of these lymphogranulomatous strictures of the rectum except a radical measure. Under this heading probably the most popular is the one-stage abdominoperineal resection of Miles. It is to be chosen for malignancy although a permanent colostomy must of necessity be established; a proctosigmoidectomy (Babcock)[103,105] performed by the abdominal or perineal route avoids this by the establishment of a functioning perineal anus. Any intra-abdominal extirpation performed carries an exceedingly high mortality; with colostomy followed by perineal excision the mortality is practically nil. Twenty-seven patients operated upon by the author[106] are living and well even though the second stage in some instances may have been stormy and the convalescence protracted.

Colostomy. Preliminary Step. A permanent left inguinal colostomy is established through a muscle-splitting incision. This incision approximately three inches in length is made with its center cutting at right angles an imaginary line drawn between the anterior superior iliac spine and the umbilicus. This places the incision parallel to the fibers of the external oblique muscle and aponeurosis. The incision is made through the skin which is retracted; bleeding points are ligated with No. 35 alloy steel wire or No. 0 plain catgut, and drapes are clamped with towel clips to the skin edges. The external oblique muscle and its aponeurotic fibers are separated in the direction of the fibers and retracted, thus exposing the internal oblique muscle. This and the transversus abdominus muscle fibers are separated and retracted in a similar fashion. The fascia transversalis, extraperitoneal fat, and peritoneum are elevated and incised parallel to the skin incision. The sigmoid colon is found readily by passing the hand into the abdomen along the lateral wall of the peritoneal cavity and then bringing it medially along the lateral leaf of the mesosigmoid. When it has been identified by its anatomical position, appendices epiploicae, haustra, and taeniae coli, the sigmoid is brought out of the wound. Any portion of the sigmoid which is normally mobile by virtue of its mesentery may be selected. With the bowel withdrawn beyond the skin margin a small incision is made perpendicular to the axis

of the gut in an avascular area of the mesosigmoid. A glass rod with rubber tubing attached to one end is passed through this opening and allowed to rest on the skin surface; after this the free end of the rubber tube is passed over the protruding bowel and slipped onto the opposite end of the glass rod. The peritoneum is now closed loosely around the bowel with No. 1 chromic catgut and the internal and external oblique muscles approximated with No. 32 alloy steel wire in an interrupted fashion. Finally, the skin is drawn together with silk or No. 35 alloy steel wire and vaseline gauze tucked snugly around the bowel.

The second step consists of opening the bowel. This may be done immediately but is usually performed between the second and fifth day. Subsequent care consists of daily dressing, cleansing the skin with alcohol, and the application of gauze saturated with mineral oil. Fluids are given by vein and skin during the first forty-eight hours to the extent of not less than 3,000 cc. daily. A liquid diet is prescribed after forty-eight hours provided distention is not marked. Milk should be avoided. Skin sutures are removed between the fifth and seventh day.

Division of the Colostomy. This may be done in the operating or patient's room. Without anesthesia the surrounding skin and suture line are protected by gauze or toweling and the protruding bowel divided transversely in its center, using a scalpel, cautery or electric knife, until the glass rod is encountered. Bleeding points are coagulated or ligated with plain chromic catgut. A finger is introduced into the lumen of the upper and lower loop and a few ounces of warm olive oil instilled. This and the administration by mouth of mineral oil are of utmost value in establishing the fecal flow. Thereafter the patient may be given a soft diet and the lower loop irrigated with warm potassium permanganate 1.8000 solution. While the patient may be discharged on the tenth day the irrigation of the lower loop should be continued for a period of three, six or even nine months which diminishes the degree of infection. The general resistance is improved by the short-circuiting process and methods toward rehabilitation: fresh air, sunshine, good wholesome food, tonics and vitamins.

Perineal Excision. Under lumbar analgesia the patient is placed in the lithotomy or inverted position. (In the case of a male an indwelling catheter is tied in the bladder.) The anus is closed with plain silk or catgut after an iodine-saturated strip of gauze is inserted into the rectum. An incision is made from a point one inch anterior to the anal verge on either side of the anus to a site overlying the coccyx tip. The incision is deepened on all sides and the deep fascia denuded. All portions of the levator ani are severed and ligated. When the lateral and posterior aspects are freed, the rectum is carefully dissected from the vagina or prostate. When the lower edge of the prostate is reached the rectum is stripped backward from it until the two seminal vesicles are seen. The stripping is continued, aided by blunt dissection with closed scissors, in order to separate the rectum from the vesicles. Following separation of the ductus deferens and vesicles from the rectum the lateral ligaments are divided and here the peritoneum comes into view. When the stricture involves the summit of the rectum, it is necessary to open the peritoneum. In this case it is nipped with scissors and freed on all sides. The mesosigmoid is divided between clamps and the vessels ligated. A site well above the stricture is selected and the peritoneal and muscular coats of the gut are incised circularly and stripped backward so as to form a wide groove. In this groove one clamp is applied below and another above, following which the bowel is divided with a cautery. The freed segment is removed from the operative fields. The remaining stump is ligated with catgut, the clamp removed, and the closed end of the gut inverted by means of a purse-string suture. Where the peritoneum is opened during the dissection, it is now closed around the

upper segment interruptedly. To avoid an attempt at closing the perineal wound by suture a large sheet of gauze is placed in the wound and loosely filled with a series of cigarette drains. These are removed after the fourth day. Patients are allowed out of bed after the fourteenth day when small hot sitz baths are begun.

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Thoracic Surgery

POSTOPERATIVE PULMONARY ATELECTASIS

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ALTHOUGH a century has passed since its first recognition and experimental production, pulmonary atelectasis still constitutes the most common pulmonary complication following intra-abdominal operations. Rink[61] (1938) recently stated that from a series of reported cases, pulmonary complications occurred in 14 per cent of the abdominal operations and in only about 1 per cent of nonabdominal operations, and that the latter as a rule were other than pulmonary atelectasis. These statistics are in accord with those of King[38] (1933) who reported an incidence of 14.3 per cent for the abdominal group and 1.2 per cent for all others. The importance of atelectasis is more appreciated when its relationship to postoperative pneumonia is considered. It is now an established fact, based on experimental and clinical evidence, that unless effective early treatment is instituted postoperative atelectasis is likely to develop into pneumonia. The proof of this statement will be presented later in the discussion dealing with pathologic physiology.

The term, atelectasis, is derived from two Greek words, "ateles," meaning imperfect, and "ektasis," meaning expansion. It was first applied by Jorg[35,16] in 1832, on ideas gained from his father, to a condition of the lungs observed in newborn infants after death. It was not recognized as a (pulmonary) complication following abdominal operations until almost seventy-five years later when Pasteur[57,58] (1908 to 1911) published a series of articles on the subject. The term, "massive collapse of the lung," was originated by Pasteur to designate a type of atelectasis which involved an entire lung lobe as compared to patchy atelectasis. This differentiation unfortunately was on an etiological basis and subsequently led to considerable confusion when attempts were made to reproduce the condition in experimental animals. To Sir James Barr[5] (1907) goes the credit of diagnosing the condition as a postoperative complication. After ruling out pleurisy with effusion by attempted thoracentesis, a diagnosis of atelectasis was made and the patient recovered "under a course of respiratory gymnastics."

ETIOLOGY

It is not the purpose of this paper to go into great detail concerning the historical background of the theories pertaining to the etiology of postoperative pulmonary atelectasis. These theories have been thoroughly reviewed by several authors including, among others, Churchill[14] (1925), Brunn and Brill[10,11] (1930), more recently by Moore[52] (1939) and will only be mentioned here in connection with various etiological factors as they are now recognized. Although the etiology of atelectasis has been debated for almost a century, a universal agreement was not forthcoming until within the past two decades. Much of the argument has been as to what factors are most important and which are secondary. The schools of thought have been divided into three groups and will be discussed as such.

1. *Bronchial Obstruction.* As early as 1853, Gairdner[26] was aware of the frequency of "collapse of the lung" and recog-

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nized both the "diffused and lobular" types. He believed that bronchial obstruction which was dependent on a number of secondary factors (i.e., mucus in the bronchi, weakness or inefficiency of the inspiratory power and inability to cough and expectorate) was the essential etiological factor in the production of atelectasis. He thought that the obstruction was of a ball-valve type and that the air was removed by way of the air passages.

Bartels[6] (1861) also favored bronchial obstruction as the primary etiological factor but agreed with Fucks[25] (1849) who was the first to point out that the entrapped air was probably absorbed by the blood. It remained for Lichtheim[44] (1878), through ingenious and painstaking experiments, to prove that the entrapped air following bronchial obstruction was absorbed by the blood. He demonstrated that when the blood vessels were tied no atelectasis occurred. In addition he determined the rate of collapse, and found that complete absorption of pure oxygen occurred from the lungs within forty-five minutes while nitrogen required at least twenty-four hours. As pointed out by Sewall[63] (1921), these experiments did not disprove the possibility of the air being removed from the lung by the ball-valve action of the bronchial plug.

Elliott and Dingley[21] (1914) in a review of eleven patients were among the first to recognize atelectasis as an important postoperative complication. A viscid mucopurulent secretion was found in all instances and bronchial obstruction was thought to be essential for the production of atelectasis, although immobility of the diaphragm was considered as a secondary factor. More recently an abundance of both clinical and experimental evidence (Jackson and Lee[34] (1925), Andrus[3] (1925), Hearn and Clerf[30] (1927), Lee et al.[40,41,42] (1928), Coryllos[18] (1930), Adams and Livingstone[1] (1931), Korol[37] (1931)) has left no doubt that bronchial obstruction is the one primary factor essential for the production of pulmonary atelectasis.

2. *Decreased Inspiratory Force.* Pasteur[55,56,59,60] (1890 to 1914) who was the first strong supporter of this theory, was much influenced by observing pulmonary atelectasis in a number of patients with postdiphtheritic paralysis of the diaphragm. He also believed that paralysis of the bronchial muscles might in some way aid in the production of atelectasis and was among the first to recognize its importance as a postoperative complication.

Briscoe[9] also believed that quiet breathing in the supine position in patients who did not fix their chest with the abdominal muscles, would normally lead to atelectasis.

Likewise Bradford[8] (1917 to 1919) after observing a number of patients with atelectasis following and on the opposite side to trivial war wounds of the chest wall, was inclined to believe that reflex immobility and retraction of the chest wall were the primary etiologic factors.

The fact that atelectasis is by far the most frequent pulmonary complication of abdominal operations (Mastics et al.[48] (1927), Scott[66] (1927)) is supportive evidence for this theory since it has been repeatedly demonstrated (Churchill and McNeil[15] (1927), Muller et al.[54] (1929), Carlson[13] (1932)) that operations, especially in the upper abdomen, reduce both vital capacity (as much as 50 to 60 per cent) and inspiratory effort to a considerable degree.

However, the fact that diminished inspiratory force due to paralysis of the diaphragm or immobilization of the chest wall is insufficient in itself to produce atelectasis is being demonstrated continually in the surgical treatment of pulmonary tuberculosis. Thus it must be regarded as contributory in nature.

3. *Nervous Reflex Contraction of the Lung.* In 1903, Dixon and Brodie[19] produced atelectasis experimentally by the direct action of drugs, and by reflex vagal stimulation causing bronchoconstriction. In order to obtain these results, artificial respiration with little force and with the expiratory phase prolonged was required. If the reverse were tried (i.e., much force

and a long inspiratory phase), distention of the lung resulted.

Scrimger[69] (1921) and Scott[65] (1925) also believed that nervous reflex production of contraction of the bronchial muscles, such as that demonstrated by Carlson and Luckhardt[12] (1920) was the major etiological factor in the evolution of atelectasis. Support for this theory is found in the report by Bergamini and Shepard[7] of two fatal cases of bilateral total atelectasis which occurred during surgery and in whom no bronchial obstruction or secretions were demonstrable at necropsy. More recently Thomas[70] (1938) has reported the experience of Mr. J. E. H. Roberts, who observed the sudden development of atelectasis through an open thorax when intratracheal aspiration was started. It was pointed out by Thomas that cases of atelectasis produced by this reflex mechanism represent a small minority, probably less than 10 per cent, the vast majority resulting from bronchial obstruction by retained secretions.

Although, as stated by Coryllos,[16] no one has given "an exact and precise description of the reflex involved or of the nature of the stimulus itself," it is yet to be proved that a small minority of cases are not the result of this mechanism.

PATHOLOGIC HISTOLOGY AND PHYSIOLOGY

Distribution. Postoperative atelectasis may vary markedly in amount and distribution. The lower lobes are more commonly involved and may present only small patches of airless parenchyma or the entire lobe may be flabby and contain no air. The process may involve all of both lower lobes as reported by Ball[4] (1928), or as is not infrequently seen may include an entire lung.

The amount and distribution of involvement may vary from hour to hour (Hearn and Clerf) either increasing or decreasing, and may shift from one side to the other by "internal drainage" (Faulkner,[24] 1932). Doubtlessly many cases with the patchy variety of atelectasis clear spontaneously by the routine changing of the patient's position in bed, with little evidence of its presence manifested. When pathogenic organisms are dammed back by the obstruction, symptoms soon appear. In 1918, Whipple[72] found that a pneumococcus of the type IV group could be cultured from most patients having this complication. Whether these are secondary invaders from the nasopharyngeal flora is not definitely known. However, most available information indicates that they are primary in nature as a result of interference with drainage of the lung.

Pathology. Since the prognosis of postoperative atelectasis is usually good, little necropsy material has been available for study. If atelectasis of a lobe is complete, the lung lobe will be markedly shrunken, flabby as a piece of muscle, of a dark reddish-purple color and will sink when placed in water. Microscopic sections reveal approximation of alveolar walls with some mucus in the bronchioles. If micro-organisms are trapped by the obstruction, leukocytes and lymphocytes may be seen in the air passages and alveoli. Later stages may show edema of the parenchyma, but it is questionable whether this does not represent the development of pneumonia. Coryllos[17] (1929) believed that not only was there a close relationship between postoperative bronchitis, atelectasis and pneumonia, but that the three conditions represented three stages of a single process. He demonstrated experimentally the similarity of the microscopic appearance and the disturbed circulation observed in atelectasis and pneumonia. Clinical experience has revealed the co-existence of the two conditions. Furthermore, differentiation between the two by means of symptoms, physical and *x*-ray signs is not infrequently very difficult especially when dealing with the patchy type of atelectasis. Thus it is obvious that when these facts are considered, postoperative atelectasis assumes a much more important rôle.

Physiologic Considerations. In the presence of patchy atelectasis without wide

distribution, little alteration of cardio-respiratory function may be effected. However, when an entire lung or only one lobe becomes entirely atelectatic, marked changes may be manifested and may vary directly with the rate of lung collapse.

Reduction in respiratory function which is already encumbered by a lowered vital capacity, is apt to produce dyspnea and hyperpnea. This is all the more likely because of the associated lowering of blood oxygen saturation. Andrus,[3] in 1925, found that thirty minutes following occlusion of the bronchus of an entire lung, the volume flow of blood through that lung was reduced to 31.0 per cent of the total cardiac output, the latter having been increased 14.0 per cent over the preobstruction level. Twenty-four hours later this was decreased to 28.0 per cent, and at the end of one month to 8.0 per cent of the preobstruction level. These results have been substantiated more recently by Moore[53] (1931) and also in our laboratory (Adams et al.,[2] Phillips et al.[60] (1940).

Another striking physiologic alteration produced by atelectasis of an entire lobe or lung, takes place in the pleural cavity. The normal intrapleural negative pressure varies in different locations of the pleural space and with the respiratory cycle. Whereas the average normal is about −6.0 mm. of mercury on expiration and −8.0 mm. of mercury on inspiration, Elkin[20] from measurements made in three clinical patients reported intrapleural pressures as high as −12.0 mm. of mercury in expiration and −15.0 mm. of mercury on inspiration on the effected side with pressures on the opposite side remaining approximately normal. This is similar to the measurements recorded in our laboratory (Escudero and Adams[22]) on dogs with unilateral collapse of an entire lung. Here the intrapleural pressures were altered from a normal of −6.0 and −8.0 cm. of water to −14.0 and −18.0 cm. water on the effected side with also a decrease in pressure on the opposite side to −8.0 and −10.0 cm. water correspondingly on expiration and inspiration. This discrepancy in pressure changes on the side opposite to that of the collapse occurring in dogs as compared to man is explained likely due to the more mobile mediastinum in the former, thus causing a greater shift of mediastinal structures toward the side of atelectasis, a finding also present to a lesser degree in children.

The rate of production of atelectasis of an entire lobe or lung has been much debated. From experimental studies as well as clinical observations it has been found to vary considerably according to the etiological factors, i.e., the type and effectiveness of bronchial obstruction and the character of the respiratory movements which are present in individual cases. Lee and his coworkers[41] (1928) produced atelectasis of an entire lung in dogs within from thirty minutes to three hours by obstructing the bronchus with either 100 per cent acacia or with bronchial secretions removed bronchoscopically from a patient having massive atelectasis of the entire left lung. The dogs were under deep narcosis but the respiratory movements became exaggerated on the side of the chest opposite to the side of collapse. Experiments performed on dogs in our laboratory[71] (1930) demonstrated conclusively that a straining or grunting type of respiration with a check-valve type of obstruction markedly shortened the time necessary for the production of atelectasis of an entire lobe or lung. Serial *x*-rays revealed marked changes present within a two-minute period. This increased rate of production seems logical since the air in the lung has two avenues of escape, i.e., through the bronchus and also by way of the blood stream. Further, in total obstruction with grunting or straining respiration, absorption of the entrapped air by the blood stream is likely to be more rapid than with quiet respiration since gas absorption varies directly with its partial pressure. In experiments performed at a later date[22] (1933) complete bronchial obstruction in the presence of a normal type of respiration without sedation or

narcosis produced total atelectasis of an entire lung lobe within a three-hour period.

CLINICAL PICTURE AND DIAGNOSIS

Postoperative atelectasis characteristically makes its appearance twenty-four to seventy-two hours after surgery but may develop as late as seven to ten days postoperatively. It may be sudden or insidious in onset and varies from mild to severe in its course. There is usually a sharp rise in temperature, pulse and respiratory rate accompanied by various degrees of dyspnea. A cough is usually present and may or may not be productive of sputum. Occasionally, a chill will accompany the rise in temperature, and in severe cases cyanosis may be present. Pain is a variable complaint and whether this represents a combination of atelectasis and pneumonia with pleurisy is not known. As a rule all of these symptoms are not present in each individual case, however, elevation of temperature, pulse and respiration are usually present.

As stated by Brunn and Brill[10] (1930), "the chief characteristic of the physical findings is their variability." They may vary from hour to hour according to the amount of involvement and the type of or absence of bronchial obstruction.

It is generally agreed that the most important finding on physical examination for making the diagnosis of atelectasis is displacement of the heart and mediastinum toward the side of involvement. Unfortunately, this is not always easy to determine in postoperative patients by physical examination and therefore portable *x*-rays are indicated when the condition is suspected. The above findings are usually accompanied by elevation of the diaphragm on the involved side with limitation of motion of the chest wall and narrowing of the interspaces, the degree of which will be somewhat determined by the amount of lung tissue involved. If the lesion is bilateral, or of the patchy variety, the above findings may be less striking or minimal in degree. In the case of patchy atelectasis physical examination may be practically normal or consist only of some diminution in breath sounds with or without the presence of râles. However, in the lobar type or when an entire lung is involved, the percussion note will be dull or flat and breath sounds will be altered. If the bronchi are completely occluded, no breath sounds or râles are demonstrable and tactile fremitus is diminished or absent. When the bronchial obstruction is released, loud moist râles and bronchial breathing are present and tactile fremitus will be restored. Many of these findings are similar to those of early pneumonia especially of the bronchial type and for this reason the lesion has undoubtedly been diagnosed as pneumonia when little shift of the heart was noticeable. From experimental work on dogs, Coryllos and Birnbaum[17] (1929) concluded that atelectasis represented a stage in the development of pneumonitis. At the present time it is generally believed from clinical observations that more serious complications such as lung abscess and pneumonia develop from atelectasis if the latter is not promptly diagnosed and properly treated. For this reason, as mentioned above, *x*-rays of the chest are frequently indispensable. Here also the evidence is not always conclusive, for patchy atelectasis has in the past been frequently diagnosed as bronchopneumonia. Other laboratory tests may be of little help in differentiation between the two conditions since the white blood cells in lobar atelectasis are usually increased to 15,000 or 20,000; and although the sputum is often tenacious in character, it may be quite thin, scanty or abundant and usually contains a pneumococcus of (one of) the type IV group.

Factors which aid most in diagnosis are: (1) awareness of incidence after abdominal operations; (2) time of onset—twenty-four to forty-eight hours after operation; (3) sudden rate of development and severity of symptoms; (4) shift of heart and mediastinum on physical and *x*-ray examination, and (5) bronchoscopic finding of bronchial obstruction.

TREATMENT

The treatment of postoperative pulmonary atelectasis may be divided into prophylactic and active. Since the incidence of atelectasis following operations can be materially reduced by attention being given to etiological factors, the importance of prophylaxis is at once apparent.

Prophylaxis. As mentioned above the primary etiological factor in most cases of atelectasis is bronchial obstruction usually produced by retained secretions. All other factors are secondary but nevertheless may be of just as much importance, and not infrequently two or more factors may be complimentary in the production of the complication.

1. *Infection.* Since this is an important factor in most pulmonary complications, adequate attention should be given to careful oral hygiene. Furthermore, all major operations of election should be delayed for some time (several weeks) when there is a suspicion of an upper respiratory infection. Placing patients in the Trendelenburg position (about 10 to 15 degrees) until consciousness returns or even as a routine in major abdominal operations will aid in the elimination of secretions from the tracheobronchial tree by gravity. The same principle holds in the frequent change of position in bed (at least every four hours) during the first four or five days following operation.

2. *Drugs.* Since some drugs such as atropine and hyoscine tend to dry secretions, making them more tenacious and difficult to eliminate, they had best be eliminated even when excessive secretions are present (Mathes and Holman[49,50] (1929, 1930) and Thomas[70] (1938)). In our experience this has definitely reduced the incidence of postoperative atelectasis.

3. *Anesthesia.* Since the incidence of this lesion is practically the same following the use of various anesthetic agents, the attention of the anesthetist should be directed toward maintainance of the cough reflex when possible and to the early return of consciousness following operation. Careful elimination of secretions both during and following operation by gravity drainage or by aspiration through a catheter or a bronchoscope is of paramount importance (Eversole[23] (1940), Mousel[51] (1940)).

4. *Operation.* It is common knowledge that this complication usually follows abdominal and pelvic operations. Thus greater care should be given to this group of patients both in the way of prophylactic treatment as well as watching for its early manifestations following operation.

5. *Excessive Secretions.* In patients with a chronic productive cough due to chronic bronchitis or bronchiectasis particular attention should be given to the elimination of the exudate (secretions) before, during and following operation. This may be accomplished by postural drainage, encouraging the patient to expectorate or by bronchoscopic aspiration prior to operation. To aid elimination by gravity during operation, the patient is placed in a Trendelenburg position by lowering the head end of the operating table to a 10 to 15 degree angle. Aspiration by catheter may be advisable during operation in some cases in which the secretions are profuse. Following operation catheter or bronchoscopic aspiration of secretions from the tracheobronchial tree should be as thorough as possible. On return to the pavilion or room repeated catheter aspirations as described by Haight[29] (1938) will do much to prevent atelectasis and pneumonia. An indication for aspiration is the presence of loud râles and ronchi produced by mucus and pus which the patient is unable to expectorate. If this can not be removed by aspiration with a catheter, a bronchoscope should be inserted and the aspiration carried out under direct vision.

6. *Diminished Respiratory Force.* Since it has been definitely proved that in abdominal operations the respiratory force and vital capacity are markedly reduced, all attempts should be made to prevent further reduction of function by abdominal binders and deep sedation. Henderson[31,32,33]

(1920 to 1929) recommended the inhalation of carbon dioxide-oxygen at intervals following operations in order to re-expand hypoventilated lung tissue. This prophylactic measure has been given an extensive trial with reports of its value being variable, Scott and Culter[67] (1928), Scott[68] (1929), Brunn[10] (1930). However, where hypoventilation is extreme as is the case following some upper abdominal operations, there is little doubt that occasional hyperventilation is beneficial.

7. *Cough.* Since coughing is probably the best natural defence against stagnation of secretions in the bronchial tree, every effort should be made to keep it effective. Due to the production of pain on straining the abdominal wall, the patient is reluctant to cough and expectorate retained intrabronchial secretions. This can be partially overcome by splinting the wound with a binder which does not restrict respiratory movements and also by the patient using his hands to support the abdomen during the act of coughing (Sommer[64] (1941)). The use of drugs for the relief of pain is important but respiratory depressants should be limited.

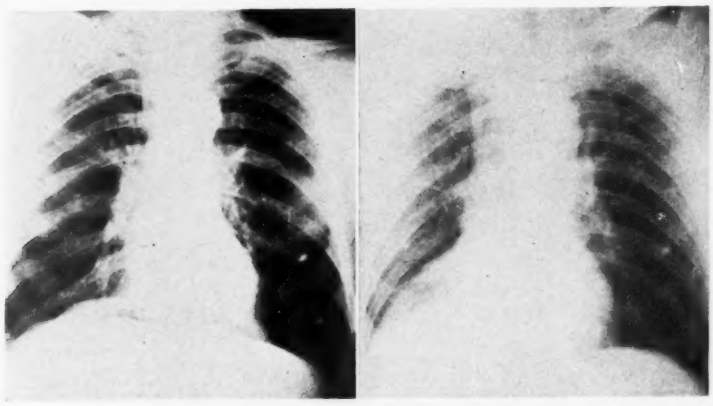
Active Treatment. When once the diagnosis of postoperative atelectasis is established, active therapeutic measures should be immediately instituted in order to obviate more serious complications.

Since bronchial obstruction due to the stagnation of secretions is present in the majority of cases, clearing of the air passages and re-expansion of the atelectatic lung is the first consideration.

The amount of lung tissue involved and the severity of the symptoms will determine to some extent the therapeutic measures indicated and the urgency of their application. Since marked hypoventilation and an ineffective cough are usually manifested, correction of these factors should be given first attention. Coughing will be more effective in a conscious patient with the head rest somewhat elevated. By supporting the wound with a binder which does not restrict respiratory activity and by splinting the abdomen with the hands, the patient may be able to raise the obstructing secretions (Saute[62] (1927)). Persistent encouragement in this regard is worth while and in the end is often rewarded. Sedation for the control of pain is helpful in producing an effective cough but respiratory depressants should be used sparingly. Recently, Guis[27,28] (1940) has made use of a paravertebral procaine block of the tenth, eleventh and twelfth dorsal and the first and second lumbar nerves for the relief of pain that interferes with cough, and for improving ventilation of the lung and increasing vital capacity following upper abdominal operations. Overbreathing or hyperventilation may also be obtained by the use of a 95 per cent oxygen and 5 per cent carbon dioxide mixture and should be administered for five minutes several times a day according to the needs of individual patients. This will not only reinflate the hypoventilated lower portion of the lungs but will increase the force and effectiveness of the cough and tend to loosen obstructing secretions.

Drainage of the bronchial tree through gravity by placing the patient in the Trendelenburg position and by change of position in bed from side to side and on the abdomen is of great value in the reinflation of an atelectatic lung. This is more effective when the secretions are thinned by the use of steam inhalations or expectorants. If the retained secretions are profuse or very tenaceous and the above measures are insufficient to clear the bronchial tree, aspiration by means of a catheter (No. 16 French urethral) inserted through the nose and directed into the air passages should be carried out without delay. The catheter may be introduced more easily with the patient in a semisitting position and passed into the trachea as the patient takes a deep breath, as described by Haight and Ransom[31a] (1941). The patient is then placed in a horizontal or slight Trendelenburg position for aspiration. By having the patient cough and repeating the suction intermittently on several occasions, effective

removal of the secretions is likely. The procedure may have to be repeated several times. If this procedure appears to be ineffective, or the severity of the symptoms seem to warrant it, bronchoscopic aspiration should be promptly carried out (Haight and Ransom[31a] Leopold[43]). This can be accomplished in the patient's room and may be a life saving measure. Bronchoscopic aspiration has the advantage over catheter aspiration in that the air passages may be more completely cleared of secretions under direct vision.



A B

Fig. 1. Case 1. A, *x*-ray of chest before operation. B, *x*-ray of chest twenty-four hours following operation.

More recently the use of chemotherapy has been found of value in the management of pulmonary atelectasis. Since bronchopneumonia may readily follow the development of atelectasis, the use of sulfapyradine or sulfathiazole during the reinflation of the collapsed lung is of real importance. The dosage used for this purpose is similar to that employed in the treatment of pneumonia.

The following cases illustrate various clinical features and therapeutic measures employed in the management of pulmonary atelectasis complicating operations:

CASE REPORTS

Case 1. A white male of fifty-six in fair general condition was admitted for treatment of carcinoma of the stomach. On August 13, 1941. a partial gastrectomy was made under ethylene-oxygen anesthesia, following M.S. Gm. 0.010 and calcium nembutal Gm. 0.27 premedication. Sedation postoperatively consisted of M.S. Gm. 0.010 and 0.015 during the first day and a similar amount the second day.

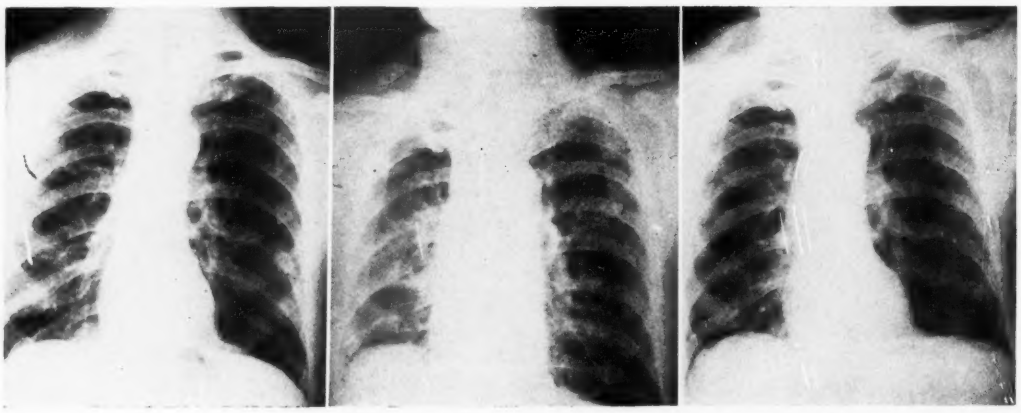
On August 14 the temperature suddenly became elevated to 104.3°F. (rectal) and the patient felt chilly; the pulse was 125, respiration thirty per minute. A cough with grayish-yellow sputum was noted. On physical examination the patient appeared somewhat cyanotic. Over the lower right chest posteriorly there was dullness with diminished breath sounds and some coarse râles. The heart was deviated markedly to the right. Atelectasis or pneumonia was suspected. The sputum contained many organisms including pneumococci. The white blood count was 13,800 (preoperative white blood count, 6,850). An *x*-ray revealed the heart and mediastinum markedly deviated to the right and a triangular shadow representing an atelectatic right lower lobe. The right intercostal spaces were narrowed and the right diaphragm was elevated. (Fig. 1B.)

Diagnosis: Massive atelectasis of the right lower lobe and patchy atelectasis of the right upper lobe.

Coughing was made more effective by elevation of the head rest. Hyperventilation was carried[10] out every thirty minutes by rebreathing in a paper bag. Steam inhalations were begun and the patient's position was changed frequently. Sodium sulfathiazole Gm. 3.0 were given intravenously and the same dose repeated three hours later.

On August 15 the patient was much improved. The highest rectal temperature was 102°F. Sulfathiazole (Gm. 3.0) was given intravenously and other therapeutic measures were continued.

On August 16 he continued to improve, rectal temperature was 100°F. Physical and fluoroscopic examination revealed the right lung to be clear. There was an uneventful convalescence thereafter and the patient was discharged thirteen days postoperatively.



A B C

FIG. 2. Case II. A, *x*-ray of chest taken one week before operation. B, *x*-ray taken on pneumonia bed on seventh postoperative day. C, *x*-ray of chest taken nineteen days following operation.

CASE II. A white male of seventy-nine entered the hospital complaining of jaundice of two weeks' duration. Three months before admission he had a severe cold which persisted for a month. His general condition was only fair, having lost twenty pounds in weight during the preceding three months. On physical examination his chest was found normal. (Fig. 2A.)

On December 27, 1939, a cholecystgastrostomy for a carcinoma of the head of the pancreas was made under ethylene-oxygen anesthesia following M.S. Gm. 0.010 premedication. Postoperative sedation consisted of M.S. Gm. 0.020 during the first day and a similar amount on the second day.

On December 29 the patient was coughing and had a rectal temperature of 103°F. Physical examination revealed dullness, decreased breath sounds and coarse râles over the midportion of the right lung posteriorly. The heart was shifted to the right. The white blood count was 13,200. The sputum contained a type XX pneumococcus.

Clinical diagnosis: Partial atelectasis of the right lung.

Hyperventilation with 90 per cent of oxygen and 10 per cent of carbon dioxide was carried out and the patient's position in bed was changed frequently. K.I. 10 ggts was administered t.i.d. to make coughing more effective. On the following day the temperature was lower and the sputum was raised more easily.

On January 3 the white blood count was 10,900. The patient continued to improve but an *x*-ray of the chest revealed a persistence of the atelectasis of the right lung with deviation of heart and mediastinum to the right. (Fig. 2B.) Sulfapyradine Gm. 1.0 was given every four hours. On January 7 the white blood count was 9,450. An *x*-ray of the chest was normal on January 15. (Fig. 2C.) The patient had an uneventful course thereafter and was discharged on January 21.

CASE III. A white male of sixteen was admitted for a plastic operation on the right mandible. He was in good general condition and on physical examination the lungs were entirely normal.

On February 22, 1941, a bone graft from the right ninth and tenth ribs to the right mandible was made under avertin and ether anesthesia, the latter being administered by intranasal catheter following induction by the drop method. No premedication was given. Postoperative sedation consisted of M.S. Gm. 0.010 on the first day, M.S. Gm. 0.010 and nembutal Gm. 0.09 the second day and M.S. Gm. 0.020, nembutal Gm. 0.09 and codeine Gm. 0.12 on the third day.

On February 24 the rectal temperature became elevated rapidly to 105°F. He became dyspneic and cyanotic; pulse was 150, respirations 35 per minute. Physical examination revealed dullness to flatness over the left chest with breath sounds almost absent and a few râles present at the apex. The heart was shifted over to the left axilla. The sputum contained

staphylococci, streptococci and pneumococci. The white blood count was 19,200. An *x*-ray of the chest revealed marked shifting of the heart and mediastinum to the left with diuseff spotty graying of the lower three-fourths of the left lung field. (Fig. 3.)

Diagnosis: Massive collapse of the left lower lobe with patchy atelectasis of the left upper lobe.

Oxygen was given by nasal catheter. Hyperventilation with oxygen and carbon dioxide was begun and the patient's position changed frequently to aid aeration of the lung by gravity drainage. Sulfathiazole Gm. 2.0 were given intravenously and followed by Gm. 1.0 every four hours. There was definite improvement on February 25. The rectal temperature was down to 103°F. and the white blood count was 15,600. The following day the temperature was normal. Physical examination revealed a normal chest and the white blood count was 9,750. An *x*-ray revealed only patchy areas of nonaerated lung with no deviation of mediastinal structures. The course was uneventful thereafter and the patient was discharged on March 5.

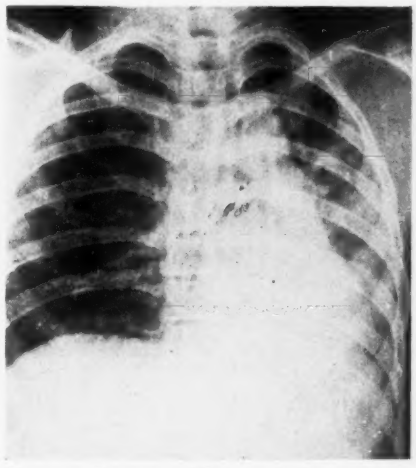
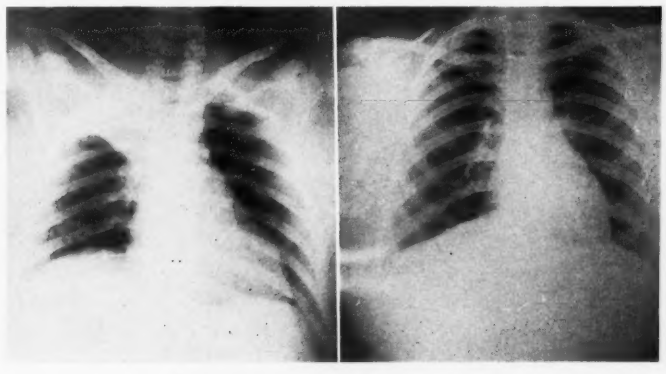


Fig. 3. Case III. *X*-ray of chest made with patient on a pneumonia bed two days following operation.

Case IV. A white female of twenty-six in good general condition was admitted for a pelvic operation. There was a history of an occasional pain in the chest with chronic wheezing.

On September 6, 1939, an incomplete hysterectomy and bilateral salpingo-oöphorectomy was made under ethylene-oxygen-ether anesthesia after M.S. Gm. 0.020 and hyoscine Gm. 0.00075 premedication given in two doses. M.S. Gm. 0.010 was given following operation to control pain.

On September 7 there was some cough with expectoration of clear mucus (no other complaints); temperature 102.2°F., pulse 130, respiration, 38 per minute. Physical examination revealed a few râles and an inspiratory stridor over the right chest anteriorly. The white blood count was 15,800 and pneumonia was suspected. The sputum contained a type 19 pneumococcus. An *x*-ray of the chest revealed an opaque shadow at the right apex, being concave inferiorly. The right diaphragm was elevated.



A B

Fig. 4. Case IV. A, *x*-ray of chest twenty-four hours following operation. B, *x*-ray of chest made thirteen days following operation.

Diagnosis: Postoperative massive atelectasis of the right upper lobe. (Fig. 4A.)

Cough was made more effective by the use of steam inhalations and K.I. 10 ggts t.i.d. Hyperventilation of the lungs was instituted and the patient's position in bed was changed frequently. Sulfapyradine Gm. 4.0 were given and followed by Gm. 1.0 every four hours.

On September 8 the temperature was only 100.4°F. and the cough was much more productive. On the following day there was marked clinical improvement; the temperature was 100°F., white blood count, 11,500. On September 10, symptoms and signs of atelectasis had disappeared, white blood count was 7,250 and sulfapyradine was discontinued. The patient's course was uneventful thereafter and she was discharged on September 18. An *x*-ray made on September 19 revealed a normal chest. (Fig. 4B.)

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DIAGNOSIS OF EMPYEMA

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IT is rapidly becoming apparent to surgeons particularly interested in the treatment of acute and chronic nontuberculous empyema that recent years have brought forth some rather remarkable developments in this field. These developments, however, have not been concerned with new or different methods of surgical treatment. Apparently, the possibilities in these fields have been almost exhausted. There has been, instead, an amazing decrease in the incidence of both acute and chronic empyema. The decrease in the incidence of acute empyema coincides with the development of the various chemotherapeutic agents for the treatment of pneumonia and the decrease has continued as the effectiveness of the various agents has become established and their use has increased. It is interesting to note that in cases in which pneumonia has been treated by specific serums, even though the pneumonia has been treated successfully, no such decrease in the incidence of postpneumonic empyema has occurred. The decreasing incidence of chronic empyema is particularly gratifying because it is due to prevention of chronicity by the early diagnosis and proper treatment of empyemas during the acute stage.

The decreased incidence of acute postpneumonic empyema resulting from the use of chemotherapy in the treatment of pneumonia has been observed too recently to be an important factor in the reduced incidence of chronic empyema. Although the use of chemotherapy in the treatment of pneumonia has resulted in a remarkable reduction in the incidence of acute empyema, it has not completely prevented the development of postpneumonic empyema. The report of the following case illustrates that pneumonia and empyema can occur while chemotherapy is being employed for another condition:

CASE 1. A woman was admitted to the hospital because of pain which had been present in the lower part of the abdomen for twenty-four hours. The pain had been accompanied by chills, nausea and vomiting. The patient also had had urinary pain and frequency for twenty-four hours before her admission to the hospital. Her temperature was 104°F. and the lower part of her abdomen was markedly tender on both sides. The uterine cervix was very tender and there was a thick yellow discharge. The diagnosis was acute pelvic inflammatory disease. Chemotherapy was instituted; a total dose of 75 to 90 gr. (5 to 6 Gm.) of sulfanilamide or sulfapyridine was administered daily for twelve days. This produced a concentration of 7.8 mg. of the drug per 100 cc. of blood. Four days after the patient was admitted to the hospital, roentgenologic examination of the thorax did not reveal any abnormality, but on the tenth day a similar examination disclosed pneumonia on the right side and fluid in the right pleural cavity. Aspiration was performed several times and Diplococcus pneumoniae was found in the aspirated fluid. On the twenty-fifth day after the patient entered the hospital an empyema was drained by means of an open operation. The recovery was complete and uneventful.

Not only has chemotherapy reduced the incidence of postpneumonic empyema but at the Mayo Clinic we have observed that when empyema does develop in spite of chemotherapy it is very different in its clinical manifestations and course from the empyema we were formerly accustomed to seeing. This will be commented on further in subsequent paragraphs.

Although the developments in the surgical treatment of empyema are most gratifying, there are many problems still remaining, as is apparent from any review of literature pertaining to the subject. It

seems likely that the diagnosis of empyema presents as many problems as any other phase of the disease.

Many terms have been applied to the condition under consideration. "Suppurative pleurisy" and "purulent pleuritis" have been suggested since these terms are descriptive of the pathologic process. "Thoracic empyema" and "pleural empyema" have been advocated since they locate the inflammatory process anatomically. "Acute nontuberculous thoracic empyema" is probably the most accurate term that can be applied, but is obviously too long for frequent usage, hence the term "acute empyema" has been generally accepted. Acute empyema can be defined as a collection of purulent fluid within the pleural cavity or interlobar spaces.

It is well known that there is a remarkable variation in the incidence of acute empyema. In general, the incidence seems to parallel the incidence of pneumonia for the year and the virulence of the invading organism prevalent at that time. Empyema occurs most frequently during the winter months. According to Nowak, in 73 per cent of the cases the disease develops during the months from December to May inclusive. It has been a generally accepted fact that empyema will develop in about 10 per cent of cases of pneumonia. In Finland's series of 3,131 cases of pneumococcic pneumonia, empyema developed in 11.7 per cent. Penberthy and Benson have reported 407 cases of empyema among 5,868 cases of pneumonia, an incidence of 7 per cent. Maes, Veal, and McFetridge reported that empyema developed in 11.2 per cent of 6,056 cases of pneumonia observed at the Charity Hospital in New Orleans in ten years. As previously stated, the use of specific serum in the treatment of pneumonia has not appreciably lessened the incidence of empyema while, on the other hand, chemotherapy has materially reduced the incidence. According to Thompson, Edwards and Hoagland, empyema developed in 10 per cent of 121 cases of pneumonia in which the patients were treated with specific sera while it developed in only four, or 2.8 per cent, of 142 cases in which the patients were treated with sulfapyridine. In a study of 351 cases of pneumococcic pneumonia, Schwartz, Flippin and Turnbull found that empyema developed in 10 per cent of cases in which specific serum was used while it developed in only 2.3 per cent of the cases in which sulfapyridine was used. Our experience at the clinic confirms these observations.

Empyema occurs more frequently among males than it does among females. According to Nowak, the incidence of empyema according to sex is almost the same as the incidence of pneumonia. This author has reported a series of 500 cases of empyema. In this series of cases the ratio of males to females was 2.3:1. Cameron found that the ratio of males to females was 2:1. In a series of 124 cases of empyema in which the patients were past ten years of age, Ochsner and Gage found that the ratio of males to females was 97:27. In a series of 122 cases of acute empyema observed at the Mayo Clinic, the ratio of males to females was 69:53. This ratio is much lower than that reported by most authors. Steinke, who reported a series of 310 cases of empyema in which the patients were children, found that 186, or 60 per cent, of the patients were boys and 124, or 40 per cent, were girls. These figures approximate those obtained at the clinic.

Table 1
Age of patients in 124 cases of empyema

	Age, Years								
	0 to 4	5 to 9	10 to 19	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79
Cases...	21	17	19	16	20	13	10	5	1

No age group is exempt from pneumonia and its chief complication, empyema. Table 1 shows the ages of patients in a series of 122 cases of empyema observed at

the clinic in a period of ten years, namely, January 1, 1931, to December 31, 1940, inclusive. In thirty-eight, or 32 per cent, of the cases the patients were children less than ten years of age. As previously stated, in all of the 310 cases reported by Steinke the patients were less than sixteen years of age. In twenty of the cases the children were less than six months of age. In 200, or approximately 66 per cent, of the 299 cases reported by Wilensky and in 32.4 per cent of the 500 cases reported by Nowak the patients were children less than ten years of age.

Empyema is rarely, if ever, a primary disease. In a great majority of cases it is caused by pneumonia or influenza. This is particularly true in cases in which the patients are children. Michalowicz reviewed 1,450 collected cases of empyema in which the patients were children. Pneumonia was the cause of the empyema in 63 per cent of these cases. In an additional 20 per cent, the empyema followed infectious diseases of childhood. Pneumonia probably was an important etiologic factor in the latter group of cases. In his review of 310 cases of empyema in which the patients were less than sixteen years of age Steinke found that pneumonia was the etiologic factor in 72.3 per cent of the cases. In eighty-nine of the 122 cases of empyema observed at the clinic, the disease followed pneumonia.

Although pneumonia is the most frequent cause of acute empyema, there are many other causes. Pulmonary abscess, bronchiectatic abscess, penetrating wounds of the thorax, pulmonary emboli, pulmonary metastasis, bronchial occlusion by tumors or inflammation, perforation of the esophagus, subdiaphragmatic abscess, or intrathoracic operations may all result in empyema. Empyema may be caused by pleurocentesis performed to aspirate fluid from the thorax, to induce a pneumothorax or to obtain material for biopsy.

Ehlers, who made a collective review of cases of nontuberculous empyema of the thorax reported in the literature, found that bacteriologic examination of the pus was made in about 3,000 cases. This examination revealed the following organisms: Diplococcus pneumoniae in 63.9 per cent, streptococci in 9.4 per cent and staphylococci in 6.5 per cent of the cases, respectively. Combinations of these organisms were not common and the Haemophilus influenzae was encountered only rarely. Other organisms that have been found rarely are Actinomyces bovis, Bacteroides funduliformis, Eberthella typhosa, Escherichia coli and Bacillus megatherium. In a series of 500 cases of empyema reported by Nowak, bacteriologic examination of the pus disclosed the following organisms: Diplococcus pneumoniae in 63.4 per cent, hemolytic streptococci in 15.6 per cent and Staphylococcus aureus in 4.8 per cent of the cases, respectively. Miscellaneous organisms were found in 9.2 per cent of the cases, and in 7.0 per cent of the cases the infection was "putrid." Steinke made a study of 310 cases of acute empyema in which the patients were children. The results of bacteriologic examination of the pus in 175 cases were recorded. Diplococcus pneumoniae was the only organism found in 69.5 per cent of these cases. Staphylococci were the only organisms found in 15 per cent of the cases and streptococci were the only organisms found in a similar percentage of the cases. The following organisms were found in the stated percentage of the 122 cases: Diplococcus pneumoniae in 60 per cent, streptococci in 28 per cent and staphylococci in 2 per cent.

The high incidence of infection of the blood stream observed by Nowak is worthy of note. Positive cultures were obtained in 120 of the 221 cases in which an attempt was made to culture organisms from the blood. The following organisms were found: Diplococcus pneumoniae in ninety cases, streptococci in sixteen cases and staphylococci in four cases.

In 47.8 per cent of the cases reported by Steinke the disease was situated on the left side of the thorax and in 43.9 per cent it

was situated on the right side. The disease was bilateral in 3.5 per cent of cases and the situation was not stated in 4.8 per cent. In the cases observed at the clinic, the site of the empyema was as follows: on the right side in seventy-four cases, on the left side in forty-six cases and bilateral in two cases. In 51.2 per cent of the cases reported by Nowak the empyema was on the right side, in 42 per cent it was on the left side, in 5.8 per cent it was bilateral and in 1 per cent it was interlobar. In a study of a series of cases collected from the literature, Ochsner and Gage found that the site of the empyema was as follows: on the right side in 435 cases, on the left side in 525 cases and bilateral in twenty-seven cases. Although the site of the empyema is of some interest, it apparently is not of great significance. According to some authors the mortality rate is higher in cases in which empyema is situated on the left side than it is in cases in which the lesion is situated on the right side, but other authors have reported opposite findings.

The diagnosis of acute empyema should not present great difficulty if empyema has been kept in mind as a possible complication, particularly if a satisfactory roentgenogram can be obtained. Because empyema is the most common complication of pneumonia, the possibility of its occurrence must be remembered even in cases in which the pneumonia is treated by chemotherapy. Roentgenologic studies of the thorax should be made at sufficiently frequent intervals during the course and convalescence of pneumonia in order to prevent the development of an unrecognized empyema.

The question as to whether empyema is metapneumonic or synpneumonic is of some significance. Metapneumonic empyema is that type which develops subsequent to the subsidence of the pneumonic process, whereas synpneumonic empyema is that type which develops concurrently with pneumonia. The metapneumonic type is usually caused by the Diplococcus pneumoniae and has a better prognosis than the synpneumonic type, which is usually of streptococcic origin.

An afebrile period of a few days after subsidence of the pneumonic process followed by recurrence of fever has been considered to be pathognomonic of empyema, particularly of the metapneumonic type. The importance of this observation has not been borne out in our experience at the clinic. This sequence of events occurred in only about 30 per cent of our series of 122 cases. In cases in which pneumonia was treated by sulfathiazole and sulfapyridine it has been an even less reliable finding since it has been our recent experience that in cases of pneumonia in which empyema develops in spite of chemotherapy there often is little or no elevation of temperature to indicate the presence of empyema. The following case report is typical of the type of empyema we have been seeing since the development of chemotherapy:

Case II. A man, thirty years of age, was referred to the clinic with a history of having had pneumonia in the left lung four weeks previously. This had been treated with sulfathiazole. Although the temperature had not risen above 100°F. at any time after chemotherapy was instituted, the patient had failed to improve satisfactorily and had complained of progressive dyspnea. When he was admitted to the hospital the temperature was 99.2°F. It did not go above this level at any time previous to operation. The leukocyte count was 13,700. Roentgenologic studies revealed fluid in the left side of the thorax, compression of the left lung to about half its normal volume, and extensive bronchopneumonia on the right side. Following aspiration of thick pus which contained Diplococcus pneumoniae, open operation for empyema was performed on the left side. A pneumococcic empyema subsequently developed on the right side and an open operation was also performed for this condition. The patient made an uneventful recovery.

It is obvious in such cases as this that it would be futile to depend upon elevation of temperature as a clue in the diagnosis of empyema.

The physical signs that are most frequently elicited by examination of the

thorax are flatness to percussion, limitation of thoracic movement, absence of breath sounds and cardiac displacement. Thoracic pain, cough and expectoration are the symptoms most frequently complained of. According to Ochsner and Gage, the signs and symptoms of empyema, expressed in order of their decreasing frequency, are as follows: (1) dullness on percussion, (2) pain, (3) cough, (4) limitation of thoracic movement, (5) absence of breath sounds (6) expectoration, and (7) cardiac displacement. In this regard, Penberthy and Benson have pointed out that physical signs and symptoms, particularly in cases in which the patients are children, are likely to be misleading.

It must be admitted that all clinical signs and symptoms are absent in some cases. In our experience at the clinic, this has been particularly true in cases in which the pneumonia has been treated by the chemotherapeutic agents.

Roentgenologic studies of the thorax will give more accurate information regarding intrathoracic conditions than any other clinical method available. They should be carried out routinely in all cases of pneumonia, both during the course of the disease and during convalescence as it is only in this way that early and accurate diagnosis of empyema can be made. Early diagnosis of empyema is extremely important since it is only by early diagnosis and treatment that satisfactory results can be obtained. Delay incurs the systemic effects of untreated infection, the accumulation of large quantities of purulent exudate in the pleural space with collapse of the lung, development of bronchopleural fistula and other complications. The seriousness of delayed diagnosis and treatment cannot be overestimated. Fitzgerald has commented on the danger to the functional value of the lung incurred by delayed drainage of acute empyema. Horine and Baker have pointed out that in their series of 103 cases of acute empyema 75 per cent of the deaths occurred in cases in which the patients had been ill for more than three weeks. Maes, Veal and McFetridge, in a review of 100 fatal cases of empyema, found that in forty cases the patients had died of toxemia and exhaustion and that in every one of the cases the empyema had been present a month or more before treatment had been instituted. In the series of 122 cases observed at the clinic, a bronchopleural fistula developed in 25.3 per cent of the cases in which more than 500 cc. of purulent exudate was allowed to accumulate in the pleural cavity before drainage was instituted, as compared to none when there was less than 500 cc. of pus in the empyema cavity.

Lateral roentgenograms of the thorax should be taken in every case in which the anteroposterior views suggest the possibility of empyema. These roentgenograms provide an accurate means of localizing the empyema so that diagnostic aspiration can be carried out without difficulty.

Occasionally, roentgenograms may be misleading. Pulmonary atelectasis should rarely cause difficulty since there is usually mediastinal and tracheal deviation toward the affected side while empyema usually pushes the mediastinum toward the opposite side. However, thickening of the pleura, pulmonary cysts that are filled with fluid, and occasionally solid tumors may produce roentgenographic signs similar to those of empyema.

Laboratory studies other than roentgenologic examination are not of great significance as regards the diagnosis of empyema. There is usually a marked elevation of the leukocyte count with a preponderance of the neutrophilic cells, but this cannot be depended upon, especially in cases in which chemotherapy has been employed.

In cases in which the physical signs and roentgenologic findings are indicative of empyema, the final diagnosis is dependent on aspiration, which should be made at the suspected site of the lesion. Aspiration not only permits accurate location of the lesion so that the most advantageous site for drainage can be chosen but, more important even than that, it can supply a

quantity of aspirated fluid for study of the physical characteristics of the pleural exudate and the nature of the infecting organism. Demonstration of the causative organism by culture of smear should be carried out in every case of empyema in order to prevent improper management of the occasional tuberculous empyema which can closely simulate pyogenic empyema. The importance of this procedure has been stressed particularly by Alexander and Kinsella. The physical characteristics of the pleural exudate are of tremendous importance in determining the proper time for drainage. A variety of methods of determining this time has been suggested. Pearse has suggested that the specific gravity of the pleural exudate should be measured and drainage delayed until the exudate has a specific gravity of about 1.035 to 1.040. Carlson has suggested that the pus may be considered thick enough for drainage when on standing the cellular sediment composes more than 90 per cent of the total volume. Fitzgerald expressed the opinion that surgical drainage should not be carried out until a positive pressure of the pus can be registered manometrically. Suzman said that if manometric readings vary with respiration the infection is widespread without localization and that drainage should be delayed until a fixed manometric pressure indicates sufficient mediastinal fixation to permit safe drainage. According to Berman, the time for drainage can be determined by roentgenoscopic examination of the thorax. All of these methods are of scientific interest but are hardly necessary in the ordinary case of empyema. The rule of thumb suggested by Graham, Singer and Ballon, that is, to delay drainage until the pleural exudate becomes "frank, creamy pus" is a sufficiently reliable guide for all practical purposes.

In the diagnosis of empyema the chief difficulties are encountered in cases in which the disease is atypical, that is, in cases in which the lesion is apical, interlobar or mediastinal and in cases in which there are small, encapsulated pockets of pus which do not communicate with the main pleural cavity. Fortunately, such cases are rare. Even in these cases, diagnosis should not prove difficult if the clinical signs and symptoms and anteroposterior and lateral roentgenograms cause one to suspect that empyema is present.

In summation, it may be said that a diagnosis of acute empyema requires (1) a suspicion of the possible presence of empyema, (2) an evaluation of the clinical course and physical signs, (3) roentgenographic studies of the thorax in both the anteroposterior and lateral positions at sufficiently frequent intervals, (4) diagnostic aspiration of the thorax at the suspected site of the abscess as indicated by the roentgenologic studies and (5) a study of the aspirated fluid as regards the nature of the infecting organisms and the physical characteristics of the pus.

It should be emphasized that although chemotherapeutic agents have materially reduced the incidence of acute empyema they have not completely prevented the development of postpneumonic empyema. It has been our observation at the clinic that in cases in which empyema has occurred in spite of chemotherapy of pneumonia the empyema has not conformed to the general textbook picture of postpneumonic empyema but has developed later in the course of the illness. Often there has been little or no elevation of temperature and the patient has had no specific symptoms except that he failed to recover his strength as rapidly as desired. The empyema develops insidiously and produces few symptoms except a gradually progressive dyspnea owing to collapse of the lung by the accumulated pleural exudate. The empyema cavity is likely to be multilocular. Cultures of the aspirated exudate are often negative for organisms. The leukocyte count may not be elevated. The reduced incidence of empyema and the changes in its character, however, are no excuse for failure to recognize the existence of the disease when it does occur. The physical and roentgenographic signs of empyema are

not influenced by chemotherapy and these examinations should never be neglected.

Hedblom has stated that empyema may be considered chronic when it shows no marked tendency to spontaneous healing or if it is still unhealed after an arbitrarily chosen period of time. Brock said that empyema may properly be called chronic when the process of obliteration of the cavity has stopped or has become so slow as to be negligible. For purposes of discussion, empyema should be considered chronic if it is known to have persisted for more than three months.

It is probable that if all the potential causes of delayed healing of empyema could be recognized and obviated during the acute stage chronic empyema would not occur. This is, of course, the goal to be sought. Prevention is by far the best treatment of chronic empyema. It must be admitted, however, that the causes of delayed healing and chronicity may be very obscure and even if recognized it may be impossible to remove them. The physician cannot be held accountable for chronicity in cases in which empyema is the result of tuberculosis, actinomycosis, neoplasm or large bronchopleural fistula. However, he should be held accountable in those cases in which chronic empyema results from failure to recognize the empyema during its acute stage and in those cases in which the chronicity is due to inadequate drainage. In the great majority of cases, chronic empyema is due to these causes. Fortunately, the decreasing incidence of chronic empyema indicates that the medical profession is becoming aware of its responsibility in these cases.

In 1923, Hedblom reported 310 cases of chronic empyema which had been observed at the clinic. I have been able to collect 346 additional cases which have been observed at the clinic prior to January 1, 1940. This makes a total of 646 cases that have been observed at the clinic prior to January 1, 1940. In the entire series of 646 cases, the ratio of males to females was almost 3:1. In 482, or 75 per cent, of the cases, the patients were between ten and forty years of age.

In this series of cases the empyema has been present for from three months to as long as twenty or more years at the time the patients presented themselves for treatment. The empyema had been present for more than six months in about 75 per cent of the cases.

Chronic nontuberculous empyema may be divided into two main groups: (1) latent empyema or empyema that has been present for months or even years without having been recognized and for which surgical drainage has not been attempted, and (2) persistent empyema or empyema which has failed to heal during the arbitrarily chosen period of three months after surgical drainage.

Latent or unrecognized empyema represents a fertile field for prevention of chronic empyema since in about a third of the cases the disease is of this type. The development of latent empyema is almost inexcusable because proper attention and treatment during and subsequent to the course of the disease, usually pneumonia, that has caused the empyema should completely prevent the development of unrecognized empyema. Too often the disease is diagnosed as unresolved pneumonia. This condition is too rare to merit serious consideration and the burden of proof of such a diagnosis should weigh heavily upon the physician making it. Careful physical examination and roentgenologic studies of the thorax in both anteroposterior and lateral positions will indicate the true state of affairs and allow accurate localization of the process so that diagnostic aspiration can be carried out without difficulty early in the course of the disease.

Failure to recognize the presence of empyema and institute early drainage is followed by extremely serious consequences. The lung is collapsed by the accumulation of pleural exudates and becomes fixed in that collapsed condition by a thickening of the portion of visceral pleura forming a part of the cavity and by

a fibrosis within the lung itself. Complete re-expansion is often impossible. Bronchial fistulas develop in a high percentage of cases, according to Hedblom, in about 28 per cent. This not only delays healing after drainage is instituted but also produces irreparable damage to the lung and bronchi and may result in a chronic bronchiectasis. The accumulated pus may burrow through the wall of the thorax and result in an empyema necessitatis or may rarely perforate into the mediastinum, through the diaphragm or even into the opposite pleural cavity. Septic absorption from the accumulated pus produces emaciation, anemia, arthritis, nephritis and osteoarthropathy. The serious results of failure to recognize and properly treat empyema in its early stages become even more apparent when one considers the prolonged illness that results, the multiple and often mutilating operations that are necessary to heal the condition, the pain and expense connected with the whole procedure and the partial disability that may remain even in the most favorable cases.

There is no excuse in the modern practice of medicine for failure to recognize the presence of empyema. It is the most common complication of pneumonia. The diagnosis is not difficult if the possibility of this complication is considered. The diagnostic aids are readily available to every physician. As has been mentioned previously, however, it is important to remember that the postpneumonic empyema that develops in spite of chemotherapy is somewhat different in its course and manifestations from that which develops in cases in which chemotherapy is not used. However, the physical and roentgenographic signs are the same in either case.

Persistent empyema or empyema that has not healed in the three months following drainage makes up two-thirds of all nontuberculous chronic empyemas according to Hedblom. Except for ruling out the possibility of tuberculosis, actinomycosis, neoplasm or complicating factors, the diagnosis presents few problems. In almost all cases there is a history of continued drainage from a thoracic sinus after drainage for empyema, or a history of healing of the sinus followed by elevation of temperature, cough and pain in the thorax which is relieved by reopening of the sinus and drainage of the empyema cavity.

It must be admitted that improper handling of the empyema during the acute stage is almost entirely responsible for the development of chronicity. That prompt, proper, adequately supervised and sufficiently prolonged treatment of acute empyema will prevent chronicity is evidenced by a report by Bohrer of 265 cases of acute empyema without development of chronicity in a single case.

The fundamental principles of the treatment of acute empyema were firmly established by the researches of the Empyema Commission in 1918 and are as true today as they were then. These principles have been affirmed repeatedly in numerous publications. The difficulties that have arisen have been due to failure to understand and apply the principles as laid down. There has been a failure to appreciate the fact that the anatomic and physiologic changes caused by acute empyema vary with the etiologic agent and that the method of treatment also should vary. It is now generally agreed that in most cases of persistent empyema the disease was treated primarily by closed methods of drainage. Ehlers has listed the most common causes of persistent empyema as (1) too early removal of the drainage tube, (2) persistence of infection because fibrin was not removed at the time of drainage, or because the tube used did not have a lumen sufficiently large to allow adequate drainage, (3) nondependent drainage, (4) the use of a drainage tube which is too long or too short, (5) the presence of a foreign body in the cavity, usually a tube or other drainage material, and (6) delayed expansion of the lung caused by thickened pleura, bronchopleural fistula or fibrosis of the lung. On consideration of these factors it immediately becomes apparent that most of

them could be considered under the single heading of inadequate drainage and that five of the six factors are at least to some extent due to closed methods of drainage. Practically all of these factors could be obviated by the use of open drainage if employed at the proper time. The fault does not lie in the closed method of drainage itself. The method was developed particularly to obviate the dangers of open pneumothorax in cases of synpneumonic empyema of streptococcic origin that were prevalent during the influenza epidemics of 1917 and 1918. It was never expected to provide adequate drainage of a well localized empyema cavity filled with masses of fibrin. Failure of the medical profession to appreciate the limitations of closed drainage led to wide adoption of the method, use of the method in cases in which it obviously was not suitable and a high incidence of persistent chronic empyema.

The decreasing incidence of chronic empyema that I have commented upon earlier is evidence that the problem is gradually approaching solution. It is to be hoped that within the next few years chronic nontuberculous empyema will be a clinical rarity.

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PRIMARY CARCINOMA OF THE LUNG*

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THE importance of primary carcinoma of the lung as a clinical problem is well demonstrated by the many excellent contributions dealing with this subject in the past few years. While there is general agreement among surgeons and radiologists that surgical extirpation of the lung (pneumonectomy) combined with the removal of the hilar lymph-nodes is the only form of curative therapy, a discussion of primary carcinoma of the lung would seem to be in order in a volume dealing with diagnosis in surgical conditions.

Carcinoma of the lung accounts for 10 per cent of all deaths due to cancer. An absolute as well as relative increase in the frequency of cancer of the lung is generally conceded.[1] Primary cancer of the lung occurs more frequently in the male. Ninety-one per cent of our last 111 cases were males. Singer's[2] total of 536 cases shows an incidence in the male of 75 per cent.

The age incidence of carcinoma of the lung is not remarkable; the greatest incidence of the disease is found in the fourth, fifth and sixth decades. The age of the patient and the sex become important factors in the diagnosis of certain polypoid bronchial tumors. Brunn and Goldman[3] have emphasized the high incidence in the female of bronchial adenoma (63 per cent were females); and, in addition, 63 per cent of the bronchial adenomas produced symptoms before the age of forty. Gebauer[4] and others have noted the difficulty in making a histologic differentiation between adenocarcinoma of the lung and bronchial adenoma; particularly is this the case when we are basing the diagnosis upon bronchoscopic biopsy material. If the patient is a female and has a polypoid bronchial tumor that has produced symptoms before the age of forty, we are inclined to make the provisional diagnosis of bronchial adenoma until we prove it otherwise. Since there is considerable doubt if bronchial adenomas ever become malignant metastasizing tumors, the importance of the age and sex factors in relation to polypoid bronchial tumors readily becomes understandable.

Primary carcinoma of the lung occurs more frequently in the right lung than the left, but this fact does not lend much weight in the diagnosis.

The early warning symptoms of carcinoma of the lung have been repeatedly reviewed and stressed by a great number of contributors to this subject. These symptoms should be well known to all doctors by this time. The important fact is that in the great majority of cases of cancer of the lung the duration of symptoms is short; and, as Overholt[5] aptly states, the disease gives quarter for only a short time in its life history and then invariably causes death. There are a few cases of cancer of the lung in which the duration of symptoms has been present for over two years. Goldman[6] collected eleven cases from our own material, and in these patients there existed clinical symptoms of the disease for over two years. Ten of these eleven carcinomas were epidermoid in type. There are, too, those carcinomas of the lung that progress to an incurable stage before any symptoms are produced; fortunately, these cases are few. The perfection of the technic for taking miniature roentgen films of the chest and the low cost per film suggests a potent means for mass examination of our male population over the age of forty. Such a program seems an ideal method to discover the silent form of cancer of the lung.

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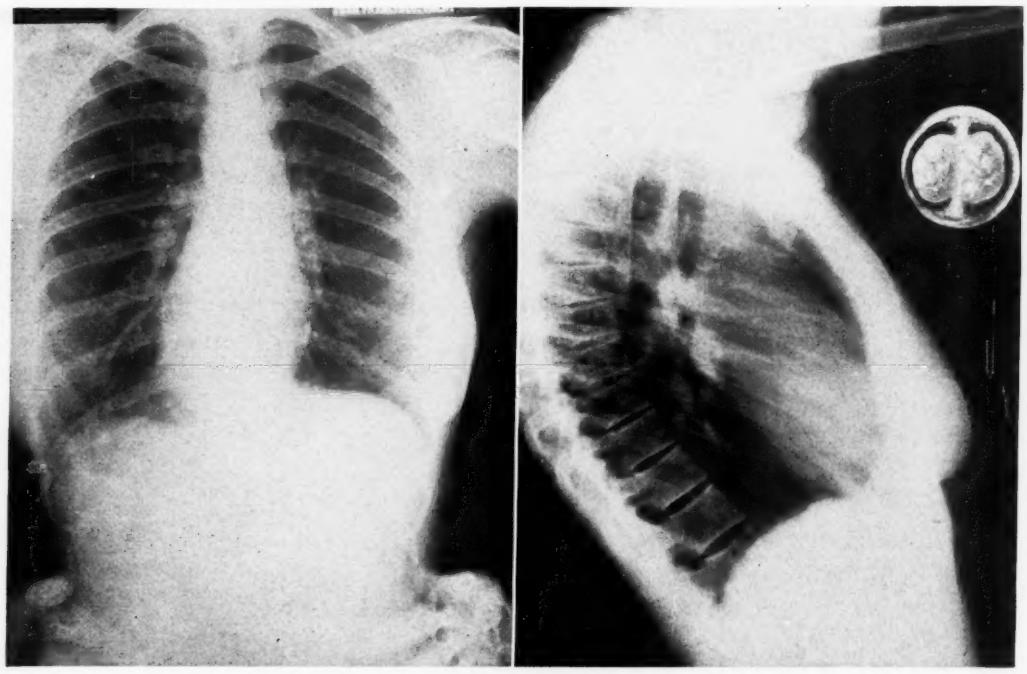
There is a truly optimistic expression which is generally present in the various studies of the problem under discussion,



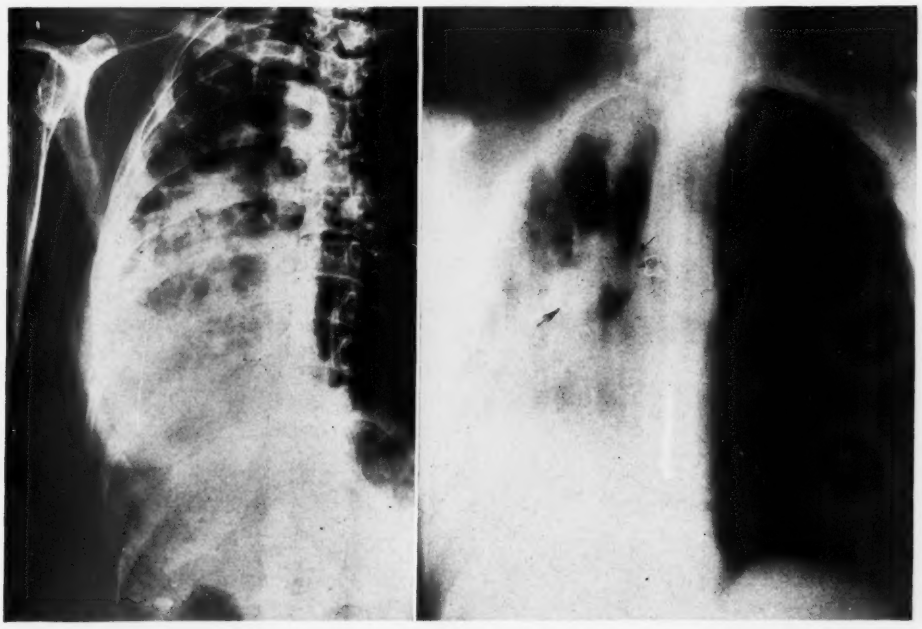
A B C

Fig. 1. The roentgen film is not infallible. This is particularly the case when an intrabronchial tumor is located near the hilar structures. A, postero-anterior roentgen film of a patient suffering from severe dyspnea. The only abnormal physical sign was slight prolongation of expiration in the region of the right upper lobe. B, lateral view of the same patient. The hilar density could be caused by enlarged lymph-nodes. C (insert upper right hand corner), artist's drawing of the large tumor (lymphoblastoma, Hodgkin's type) almost completely blocking both right and left stem bronchi in the region of the carina. The obstructive emphysema produced by the tumor is seen in roentgen film A, but there is no roentgen evidence of the presence of an intrabronchial tumor. A tomograph would have demonstrated this tumor. (Fig. 2B.)

namely, carcinoma of the lung in the great majority of cases produces symptoms sufficiently early to permit diagnosis and successful treatment provided the profession does not procrastinate. Delay on the part of the doctor must be eliminated by the acceptance of thoracic exploration for all cases suspected of harboring a cancer of the lung, provided there is not unequivocal evidence of inoperable spread and the patient is a suitable operative risk.

It is pertinent to remember there is no group of symptoms, either subjective or objective, that are diagnostic of carcinoma of the lung. Cough is the most frequent early symptom. It is customarily dry and harassing in the early stages. Later, blood-streaked sputum may accompany the cough, and upon occasion frank hemoptysis may occur. The latter symptom unfortunately is a late one. I say unfortunately because frank hemoptysis brings the patient immediately to the doctor, and roentgen films of the chest are routinely obtained of necessity. As the tumor enlarges, and if it is located in a bronchus of sufficient size, symptoms will be produced that are the direct result of bronchial obstruction. Wheezing or asthma-like symptoms may become evident. Infection in the lung parenchyma distal to the obstructing neoplasm will result in chest pains, fever and debility. When the tumor arises out in the periphery of the lung, chest pain may be the initial symptom. This pain is often the result of irritation of the parietal pleura from the adjacent parenchymal tumor. Cough, thoracic pain and hemoptysis are the cardinal symptoms of carcinoma of the

lung. When these symptoms are present in a patient of forty years or over, particularly a male, the presumptive diagnosis must be carcinoma of the lung until proved otherwise. This dictum must be strictly followed if we are successfully to treat primary carcinoma of the lung.



A B

FIG. 2. Tomographic studies. A, hard shot; postero-anterior film. Note the cystic whorls throughout the right lung field and the absence of any definite tumor mass. B, postero-anterior view 8 cm. from the back in the same patient as A. The double arrow points to the large endobronchial portion of the tumor in the right stem bronchus; the single arrows show the extrabronchial lobar extensions. This tumor was a benign bronchial adenoma.

DIAGNOSTIC METHODS

The roentgen film (Fig. 1A, B and C) comprises our first line of defense in the diagnosis of pulmonary neoplasm. The physical findings in the chest are notoriously meager in the early carcinoma of the lung; this is particularly the case when the tumor arises out in the periphery of the lung parenchyma. A small tumor located in the peripheral areas of the lung, some distance from a bronchus of any size, will cast a suspicious density in the roentgen film. When the lumen of the larger bronchi becomes involved by a malignant growth, then atelectasis of the distal lung will create an additional density that often overshadows the tumor density. Chronic pulmonary suppuration or areas of pneumonitis distal to the obstructing bronchial tumor will produce this same overshadowing effect. Body-section radiography (Fig. 2A and B) has produced the means for the roentgen demonstration of a tumor density that cannot be seen in the ordinary roentgen film.

Serial selective bronchography[7] (Fig. 3) under fluoroscopic control can be employed to demonstrate bronchial defects that are too far distal for bronchoscopic visualization. This method of roentgen examination, using an opaque oil for a contrast medium, can be used to provide additional evidence of bronchial pathology when the routine films of the chest are not conclusive.

It is our practice occasionally to introduce an artificial pneumothorax in order better to outline a suspicious shadow in the roentgen film of the thorax. It is our belief that pneumothorax will sharpen the outlines of a density, especially when the shadow appears in the lung parenchyma. This method of examination using stereo-

scopic roentgen films has been of aid to us in differentiating parenchymal tumors from areas of parenchymal inflammation.

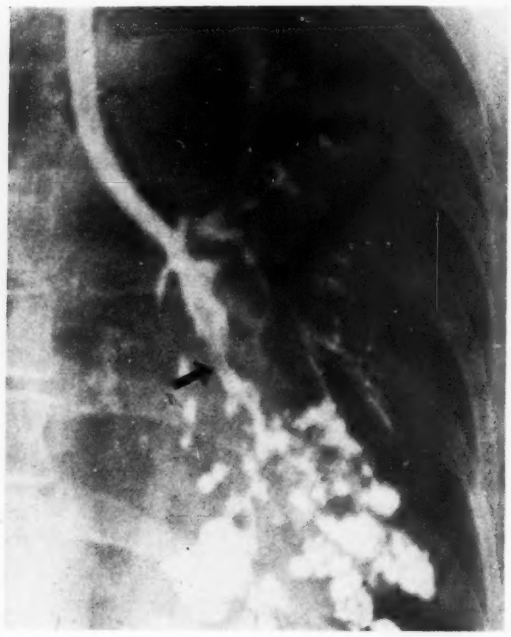


Fig. 3. Serial selective bronchography. Postero-anterior roentgen film of the left thorax. The arrow points to a filling defect in the left lower lobe bronchus. This defect was produced by an intrabronchial tumor. The saccular bronchiectasis is well demonstrated in the left lower lobe distal to the bronchial obstruction.

The statement has been made many times that 75 per cent of the patients suffering from primary carcinoma of the lung will show positive evidence of this pathology when examined by the bronchoscope. Gebauer[4] has concluded in a recent report that bronchoscopy will be negative in 40 to 50 per cent of the cases if this examination is performed at the onset of symptoms. The value of bronchoscopy is not meant to be lessened by Gebauer's conclusion, but rather this statement implies that bronchoscopic visibility of the tumor is delayed positive evidence in over 25 per cent of the cases. When the bronchoscopic examination is negative, other diagnostic methods must be employed if carcinoma of the lung is to be recognized early.

We are not interested in the late bronchoscopic picture of carcinoma of the lung in this discussion. The widening and fixation of the carina, pressure deformity of the trachea above the carina, or ulceration in a stem bronchus extending up and involving the mucosa at the bifurcation of the trachea, constitute visual evidence of inoperable carcinoma. When there is no endobronchial tumor visible, nor any break or ulceration of the bronchial wall, the bronchoscopist should look for deformity of a given bronchial opening that results from lobular atelectasis. In this latter instance, the bronchial opening may be displaced upward and lateral so that, in the case of the upper lobe, the lobar bronchial opening may not be visible through the bronchoscope. Bronchial fixation and ridging of the mucosa are suggestive findings and require further diagnostic investigation.

Examination of the sputum for malignant tissue or examination of material obtained by needle puncture of the lung have not been used with any degree of regularity in our clinic. In the former instance, Overholt[8] expresses the inclination that cancer cells will probably only become visible in the sputum in the late cases when the tumor is already inoperable or is visible through the bronchoscope. Overholt[8] and Churchill[9] believe that needle biopsy is permissible in cases that are clearly inoperable, but in those cases that may be operable they advise exploratory thoracotomy. Our practice has been very much the same as that advised by Churchill and Overholt. When pleural fluid is present, we routinely aspirate the fluid and examine the sediment by the Mandelbaum method. The fluid is usually replaced by air, and a thorascopic examination of the pleural cavity follows the aspiration of the fluid. It is regrettable that these examinations usually yield positive evidence only when the carcinoma is hopelessly inoperable.

DIAGNOSIS BY PATHOLOGIC CLINICAL DIFFERENTIATION

Koletsky[10] and Gebauer[4] have introduced a classification of primary carcinoma of the lung according to the cell type and the clinical course of the carcinoma. They

believe small-cell carcinoma, adenocarcinoma and squamous cell carcinoma are three fundamental types of bronchiogenic cancer. Late in the disease these investigators were able to distinguish these three types of primary carcinoma of the lung in 60 per cent of the cases by combined clinical, radiologic and bronchoscopic investigations.

Of the three types of carcinoma of the lung, the squamous cell cancer is undoubtedly the best suited for surgical removal, and fortunately is the most common of the three types. The relatively slow growth and mild metastatic tendencies favor the possibility of complete removal. Necrosis and cavitation are common in the squamous cell tumor, and frequently render impossible a differential diagnosis from pulmonary abscess.

Graham[11] cannot agree with Koletsky and Gebauer in that he is unable to separate small cell carcinoma from adenocarcinoma as two distinct types of cancer of the lung. There is general agreement among all doctors interested in this subject that the squamous cell tumor is a distinct type and the most favorable from the standpoint of complete removal by surgical interference.

We have not reviewed our own material in an attempt to apply Gebauer's classification and to attest our ability to use this classification to the same advantage as has Gebauer. A pathologicoclinical differentiation in cancer of the lung would seem to be a definite step in the right direction. It may be that in the future, data will be available upon which we can base the correct treatment according to the type of cancer of the lung. This would seem the logical foundation upon which to base future therapeutic improvements.

DIFFERENTIAL DIAGNOSIS

Pulmonary tuberculosis, nonspecific pulmonary abscess, lipoid pneumonia, bronchial adenoma and metastatic pulmonary tumors are the more common diseases that simulate primary carcinoma of the lung. A mistaken diagnosis of tuberculosis or lung abscess often results in the delay of proper therapy. Lipoid pneumonia and bronchial adenoma are frequently misdiagnosed carcinoma, and metastatic pulmonary tumors may be mistaken for primary lung tumors.

One must keep in mind that pulmonary tuberculosis and primary carcinoma of the lung may be present at the same time in the same patient. In addition, primary carcinoma of the lung may closely simulate pulmonary tuberculosis in its mode of onset and radiographic appearance. Pulmonary tuberculosis, on the other hand, may produce a conglomerate nodular tumor in the lung (tuberculoma) that cannot be differentiated from carcinoma except by microscopic studies of the resected specimen.

The majority of the errors in this group have been in mistaking primary carcinoma of the lung for pulmonary tuberculosis. The histories of these patients reveal that the patients were confined to bed rest for many months because the lesion in the lung suggested tuberculosis, yet the tubercle bacillus was never found in the sputum. Often the roentgenologist is responsible for this error; he looks at the roentgen films and calls the lesion tuberculosis. We have made the mistake of accepting the radiologist's report without continued follow-up effort to prove or disprove the radiologist's impression.

Tuberculomas of the lung may exist without the evidence of a positive sputum. A strongly positive tuberculin reaction may give one a clue as to the possible etiology of the nodular lobar density present in the roentgen film. It has already been stated that many times the true diagnosis is not determined until the histologic examination has been completed. Our experience in the surgical treatment of tuberculoma of the lung by lobectomy has been very meager, but a good result can be anticipated if there is no clinical evidence of activity of the tuberculous focus.

Nonspecific abscess of the lung frequently accompanies carcinoma of the lung, particularly the squamous cell type. In some cases it will be impossible to deter-

mine if a carcinoma of the lung is responsible for the abscess until surgical exploration has been carried out. Particularly is this the case when the bronchoscopic examination is negative. The best aid in the differentiation of these two pulmonary diseases is the recognition upon the part of the doctor that squamous cell carcinoma frequently produces an abscess in the lung. Because the squamous cell carcinoma is slow in growth, an abscess may form in the lung distal to the bronchial obstruction produced by the tumor. Singer[2] has stated the case clearly in these words: "The clinical picture of bronchiogenic carcinoma is frequently masked by the complications. Bronchoscopy should be a routine procedure in every case of pulmonary abscess, and the bronchoscopy should precede any type of specific treatment. When surgical drainage is carried out in the treatment of an abscess of the lung, a specimen of the abscess wall should be subjected to microscopic examination routinely. There are many cases on record of primary removal of a lung harboring a carcinomatous abscess with recovery. It is preferable to establish bronchial drainage before exploratory thoracotomy, if possible. If one constantly keeps in mind that carcinoma of the lung is often the cause of an abscess in the lung, many of the cases in this group will be explored while they are still operable.

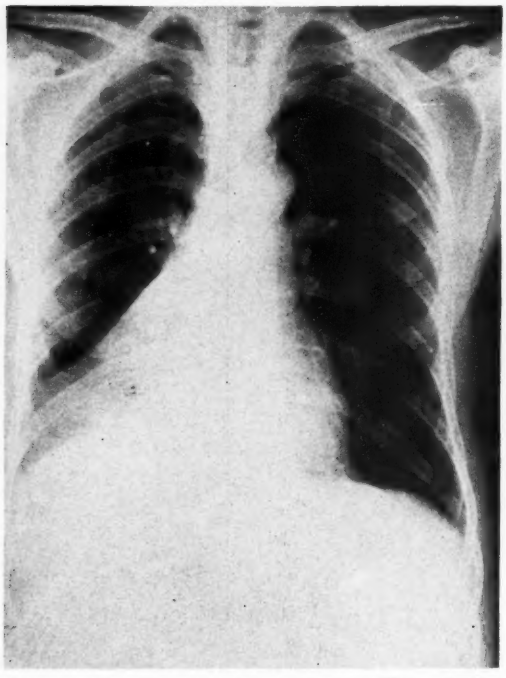


Fig. 4. Postero-anterior roentgen film of the chest showing atelectasis of the right lower lobe. Such a roentgen picture is common in carcinoma of the lung. The atelectatic lower lobe in this case was produced by granulation tissue filling the right lower lobe opening. This is a proved case of lipoid pneumonia. The treatment in this case was surgical removal of the right lower lobe.

Lipoid pneumonia (Fig. 4) has mimicked primary carcinoma of the lung several times in our experience. Lobar atelectasis, associated with a bleeding, granular bronchial obstruction and infection in the lung distal to the obstruction can all be the result of lipoid irritation. When the bronchoscopic biopsy is negative for cancer cells in such a case as outlined above, histologic search for the characteristic foam cells and appropriate lipoid stains are in order. Lobectomy or pneumonectomy may be required in the treatment of lipoid pneumonia; if the correct diagnosis can be established before operation, one is better able to determine the type of pulmonary resection necessary.

Bronchial adenoma has been mentioned before under the age incidence of carcinoma of the lung. Many excellent contributions have appeared in the recent literature describing this tumor. Goldman,[12] from our own clinic, has added considerable to our knowledge of bronchial adenoma. The tumor we describe as bronchial adenoma has been benign in all the cases we have studied to date. We are of the opinion that bronchial adenomas are definitely not carcinomas of the lung, and are anxious that they (bronchial adenomas) are not classified as carcinomas. Surgical removal of the adenoma will be necessary in many instances when this tumor is present. In our own experience, lobectomy or pneumonectomy will be the procedure of choice depending upon the location of the intrabronchial portion of the adenoma.

Metastatic tumors of the lung offer difficulties in differentiation from primary carcinoma of the lung when the tumor is beyond the range of the bronchoscope and the tumor represents a single metastatic deposit in the lung. Barney and Churchill[13] reported the successful removal of a "solitary" pulmonary metastasis from a primary kidney tumor. The patient was alive and well five years after the removal of the metastatic tumor in the lung. If a single tumor is discovered out in the peripheral lung field and a malignant tumor has been removed elsewhere in the body at some previous time, a possible secondary deposit in the lung of the original tumor should be the reasonable deduction. When careful examination does not reveal tumor deposits elsewhere in the body, exploratory thoracotomy is a sound procedure. Churchill[9] recommends the removal of peripheral nodules *en bloc* and the resection of larger growths by lobectomy or pneumonectomy with intrahilar or mediastinal dissection.

The actual surgical technic in the treatment of carcinoma of the lung is not considered to be within the scope of this communication. The operation of total pneumonectomy with mediastinal dissection technic and the removal of the mediastinal lymph-nodes has become the generally accepted operative procedure for primary carcinoma of the lung. The significant advances in the administration of intratracheal anesthesia and the utilization of cyclopropane gas have lessened the dangers of major intrathoracic operations considerably. The earlier recognition of carcinoma of the lung and the increasing experience of thoracic surgeons should show a progressive decline in the operative mortality and a progressive increase in the salvage rate for this disease.

Overholt[5] reports a salvage rate of 11 per cent (thirteen patients) and an operative mortality rate of 29 per cent (nine patients) in 127 cases of verified primary carcinoma of the lung. Thirty-one of the forty-seven cases subjected to exploratory thoracotomy were found operable. Churchill[9] notes a salvage rate of 3.2 per cent (five patients) and a hospital mortality rate of 55.5 per cent (fifteen patients) in 155 cases of primary carcinoma of the lung. Twenty-seven of the fifty-two cases subjected to exploratory thoracotomy were found operable. Churchill's figures for hospital deaths in the operation of lobectomy are probably better than those of any other clinic in this country or abroad. In 172 cases of lobectomy for various pulmonary diseases including cancer of the lung, Churchill reports nine hospital deaths, a hospital mortality rate of 5.2 per cent.

These figures are cited to show that there is still much to be accomplished to improve our present results in the treatment of carcinoma of the lung. Churchill's figures reveal that only 17 per cent of the total group of 155 cases were considered operable.

SUMMARY

1. Primary cancer of the lung is a frequently occurring tumor and is particularly common in the male.

2. Bronchoscopy will be negative in approximately 40 per cent of the cases of early carcinoma of the lung.

3. Mass examination of our male population over the age of forty, using the miniature roentgen film technic, is suggested as a means to diagnose early cancer of the lung.

4. Pulmonary symptoms appearing in a male forty years of age or older should be considered due to carcinoma of the lung until proved otherwise.

5. Exploratory thoracotomy is a safe surgical procedure and should be the method of choice to establish the diagnosis when the other diagnostic procedures are equivocal.

6. Pneumonectomy, with removal of the mediastinal lymph-nodes, is the only treatment for early carcinoma of the lung.

7. The present results in the treatment of cancer of the lung can be greatly improved by earlier diagnosis of the disease.

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There is probably no symptom, except pain, which has a wider significance than dyspnea or shortness of breath. It may be defined as a conscious desire for increased respiratory effort of pulmonary ventilation.

The brief excerpts in this issue have been taken from "Symptoms in Diagnosis" by Jonathan Campbell Meakins (Little, Brown and Company).

Genitourinary Surgery

RENAL DISEASE AS A FACTOR IN HYPERTENSION

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IT has long been known that hypertension is related in some way to pathologic change in the kidney. Some observers have claimed that renal disease is present in every case of hypertension. Others have held the opinion that so-called essential hypertension in most cases is caused by undetermined factors other than renal. Although previous investigations had indicated the relation between renal disease and hypertension, the simple experiments performed by Goldblatt and his associates[3] demonstrated this more clearly than any previous experiments. Their experiments showed that impairment of renal circulation, even when unilateral, could be an etiologic factor of hypertension. No physiologic investigation ever has received more definite clinical corroboration. Many case reports have been published which should remove any doubt that hypertension may result from certain types of unilateral renal pathologic changes. These clinical reports also have shown that elevated blood pressure may return to normal and remain so after removal of a kidney in which there is so-called surgical disease.

Not only have the experiments of Goldblatt and his associates and subsequent clinical investigations led to important therapeutic results but, what is of equal importance, they have initiated far-reaching discoveries which eventually may lead to the cause and cure of hypertension. The investigations of Page and his collaborators[4] in this field have been outstanding. These workers have demonstrated that a vasopressor substance which they have called "angiotonin" is liberated in increased amounts in the renal tissues of hypertensive animals. They have expressed the belief that angiotonin is formed by the enzymatic reaction of renin with a renin activator. Angiotonin causes increase of arterial pressure and decrease of renal blood flow as the result of arteriolar vasoconstriction. In a recent contribution Corcoran and Page[2] made the following statement: "The release of renin which initiates the process appears not to depend on renal ischemia but rather on a change from pulsatile to continuous blood flow within the kidney; that is an intrarenal reduction in blood pressure." Page and his co-workers have also shown that an extractable antipressor substance is present in normal kidneys. They recently have prepared an antipressor substance from normal kidneys which has lowered the blood pressure of patients suffering from severe, fixed hypertension.

Wakerlin and his colleagues[5] recently have demonstrated that hog renin can be used successfully in reducing high blood pressure of dogs. Not only have they been able to cause a reduction of the blood pressure of hypertensive animals by injection of renin but they have also been able to prevent hypertension in dogs by vaccination with renin. They asserted that hog renin reduces the blood pressure of dogs by stimulating the dog's own production of antirenin. They stated that the reduction of blood pressure by hog renin is not, in their opinion, due to the presence in the kidneys of a substance that reduces blood pressure but that the substance is produced in vari-

ous tissues of the body. Although much progress already has been made, it is evident that the last word has not been said in this fascinating search for the etiologic factors involved in hypertension.

After this brief review of recent investigation, let us now consider an etiologic factor of hypertension which has been definitely proved by clinical experience, namely, the so-called surgical kidney. The first question which arises is, how often are patients observed who have a unilateral surgical kidney which causes hypertension? Several clinical reports have been made which grossly exaggerate the incidence of surgical kidney as the etiologic factor in hypertension. A review of cases of hypertension observed at the Mayo Clinic showed that the percentage of patients having clinical or roentgenographic evidence of unilateral "surgical" or non-nephritic renal disease is very small. In the course of routine examination of patients at the clinic hypertension was discovered in approximately 4,000 patients in one year. Clinical evidence of a non-nephritic renal lesion was found in approximately 100 of these hypertensive patients (2.5 per cent). This group of patients was subjected to careful roentgenographic, urologic and clinical examinations and only nineteen (less than 0.5 per cent of those who had hypertension) were selected for operation. In the other eighty-one cases either the renal lesion was regarded as coincident and not related to the hypertension or there were factors which contraindicated operation. These statistics would indicate that only a fraction of 1 per cent of hypertensive patients is amenable to renal surgical treatment.

The question might be asked, how is one to recognize the presence of surgical renal lesions which could cause hypertension? Is one justified in making a roentgenographic and urographic examination, and possibly a cystoscopic examination, in every case in which hypertension is present? We have found that it is entirely unnecessary to do so. It has been our experience that a surgical renal lesion is seldom present unless at least one of the following conditions is noted: (1) a history of present or previous symptoms indicating involvement of the urinary tract, or (2) the presence of pus or red blood cells in the urine, or (3) evidence of renal pathologic changes in the plain roentgenogram, or (4) positive findings on physical examination suggestive of renal involvement. Excretory urography or cystoscopic examination seldom is indicated unless at least one of the foregoing conditions is present.

It would be simple enough to make a plain roentgenogram of the urinary tract of every patient who has hypertension. The data derived from visualization of the renal outlines in the plain roentgenogram offer a valuable method of detecting surgical lesions of the kidney. A marked difference in the size and shape of the two kidneys should call attention at once to the possibility of a renal lesion. In fact, the diagnosis of atrophic pyelonephritis can be inferred in most cases from an examination of the renal outline. The outline of a small atrophied kidney with median displacement can be seen on one side, with an hypertrophied kidney on the other side.

In the routine urographic study of cases of hypertension, evidence of abnormality often is observed, such as moderate ptosis, angulation of the ureter, slight pyelectasis or slight abnormality of the outline of the calices, pelvis or ureter resulting from previous infection, pregnancy and so forth. Most of these lesions, however, usually are of little or no significance as far as hypertension is concerned. The clinical importance of many of these deformities has been misinterpreted and exaggerated in several articles dealing with this problem. It is only by careful cystoscopic and urologic examination in experienced hands that clinical evaluation of many of the apparent urographc lesions is possible.

When one has found some evidence suggestive of involvement of the kidney, the next step is to determine whether or not renal operation is indicated. The small

group of cases of hypertension in which there is clinical evidence of non-nephritic lesions will include many cases in which operation would be inadvisable for the following reasons: (1) In order to be amenable to surgical procedure, the lesion must be unilateral; evidence of bilateral renal disease would exclude a number of cases immediately from further consideration. (2) Many patients are observed who have a minor degree of unilateral renal disease which very evidently is incidental and not the cause of hypertension. It may be difficult to determine on clinical examination whether a renal lesion is a direct factor or only incidental to the hypertension. In some cases renal surgical intervention might be justifiable on a chance. (3) Patients are observed who have hypertension associated with a surgical renal lesion which may have existed so long that vascular changes and involvement of other organs have occurred to such an extent that removal of the diseased kidney would be inadvisable. (4) Patients more than fifty years of age seldom are benefited unless the hypertension is of recent onset. For these various reasons most of the patients having renal lesions who are first selected are eliminated and as a result it has been our experience that only a fraction of 1 per cent of cases of hypertension remains in which renal operation will offer a chance for relief.

What are the "surgical" or non-nephritic lesions which cause hypertension? In order of occurrence the following types of such renal lesions were observed in our series of cases: (1) chronic and atrophic forms of unilateral pyelonephritis, including those developing after previous conservative renal operations; (2) renal neoplasm; (3) renal lithiasis; (4) hydronephrosis; (5) tuberculosis, and (6) polycystic kidneys. There are other "surgical" lesions involving the kidney that have been observed with hypertension but the lesions cited are seen most often. We have found only two cases in which pressure on or obliteration of the renal artery was the apparent cause of hypertension. Acute renal infection seldom causes hypertension. That chronic renal infection is a common factor is shown in the higher incidence of hypertension in cases of renal stone and hydronephrosis in which infection occurred. It should be remembered that although the infection may be dormant, changes affecting intrarenal circulation in the renal tissues may result from a previous infection.

When one has decided on renal operation, what permanent benefits can be expected? Most observers are agreed that the best results from renal surgical intervention are obtained among young patients and when the hypertension is of comparatively recent origin. Chute[1] stated it very well when he said that only cases should be selected in which "the cement has not set" and vascular changes have not become permanently established. Permanent recovery was noted in only one of our patients more than fifty years of age. Retinal disease, when far advanced, usually contraindicates operation in adults, but may not do so in children. Another disappointing result is the fact that although the blood pressure may drop after operation, it frequently returns to its former level after a variable period, even as long as a year later. Even in a carefully selected group of patients who have surgical renal lesions, a permanent drop in blood pressure after operation is observed in only about a third of the cases. In other words, the hypertensive patient who has been selected for renal operation should be told at the outset that he has about one chance in three for recovery.

The best results have been obtained in cases of cicatricial renal infection and atrophy such as occur with atrophic forms of pyelonephritis, or after previous conservative renal operation. Approximately 60 per cent of such patients will recover. Approximately 50 per cent of patients who have hypertension and unilateral renal tuberculosis will recover after nephrectomy. The blood pressure will return to normal in about 25 per cent of cases of hyperten-

sion with renal stone or hydronephrosis. More recently in some of the borderline cases in which the hypertension was regarded as being of the so-called essential type, splanchnicectomy was performed at the time of nephrectomy. Surgical removal of a diseased kidney which seemed to be the etiologic factor of hypertension did not cause any change in the blood pressure in many cases.

The question may well be raised, is there any particular type of renal pathologic change which favors the secretion of a pressor substance? It apparently is established that intrarenal vascular pathologic change is the essential factor which causes hypertension. Although regions of tissue atrophy with vascular sclerosis of variable extent usually are present, no specific lesion in the renal tissues has been described which always is associated with hypertension. Gross and microscopic examination of kidneys removed from patients who have hypertension may show pathologic changes which are exactly similar to those found in patients who have no elevation of blood pressure. The degree of hypertension is dependent, not on the extent of renal destruction but, as recent investigation would show, on the resulting change in intrarenal blood flow. It is evident that the problems involved are physiologic as well as pathologic in origin.

CHRONIC UNILATERAL PYELONEPHRITIS

Chronic infection with cicatricial regions in the renal parenchyma is the nonnephritic renal lesion most frequently observed with hypertension. Acute renal and perirenal infection seldom is an etiologic factor. The chronic renal infection may be either primary or secondary to some other intrarenal lesion such as lithiasis or hydronephrosis. Although primary chronic unilateral pyelonephritis is not a common lesion, yet when present it frequently causes hypertension. It usually is observed in one of three forms, namely (1) unilateral diffuse pyelonephritis with only moderate reduction in size of the kidney and with limited cicatricial alterations; (2) unilateral atrophic pyelonephritis, with advanced atrophy and diffuse cicatricial changes, or (3) postoperative pyelonephritis developing in a kidney previously operated upon.

Atrophic Pyelonephritis. Because the relative incidence of hypertension among patients who have atrophic pyelonephritis is high (47 per cent), this lesion often is cited as the renal lesion which usually causes hypertension. It also is often referred to because of the large proportion of cases in which a permanent reduction of blood pressure follows nephrectomy (70 per cent). However, atrophic pyelonephritis as a clinical entity is not observed very often, as shown by the fact that operation was performed in only forty-three cases at the Mayo Clinic during a ten-year period. This lesion is the result of extensive renal infection and is characterized by widespread atrophy of the renal tissues which causes intrarenal vascular imbalance. Atrophic pyelonephritis should be differentiated from renal hypoplasia. The latter condition is characterized by a similar reduction in size of the kidney but not by the cicatricial and inflammatory changes of atrophic pyelonephritis. The urographic data usually will permit clinical differentiation. The irregular pyelectasis and caliectasis observed with atrophic pyelonephritis usually are in marked contrast to the smooth, infantile outline of pelvis and calices observed with hypoplasia. Hypertension seldom complicates renal hypoplasia.

RENAL TUMOR

The relative incidence of hypertension among patients who have renal tumor ranks next to that occurring with atrophic pyelonephritis. It is evident that the age factor must be considered in this group, since eighty-seven, or 63.5 per cent, of these patients were fifty years of age or more. It should be stated that the blood pressure was elevated only moderately (160 mm. or less) in nineteen, or 50 per cent, of the thirty-eight cases studied. Since the blood

pressure returned to normal after nephrectomy in many of these cases, it is possible that an etiologic pressor substance is secreted in the uninvolved portion of the kidney as the result of an abnormal vascular supply caused by intrarenal pressure from the neoplasm. The hypothesis of a pressor substance secreted by the tumor tissue itself apparently has been excluded by the failure to find evidence of such substances in the tumor tissue.

RENAL CALCULUS

It may be inferred that renal calculus is not necessarily an etiologic factor of hypertension, since the incidence of hypertension was found to be no greater among patients who had renal calculus than among an equal number of patients taken at random from the clinic's registration. When, however, infection develops, with subsequent intrarenal vascular imbalance, the incidence of hypertension rises abruptly. Hypertension was observed in only 3 per cent of the cases in which there was renal stone but not infection, while in cases in which there was infection it occurred in 37 per cent. The number of pus cells found in the urine in no way indicated the presence of hypertension. Hypertension occurred as often with comparatively few pus cells present as with cloudy urine. In other words, it is apparently the extent to which tissue changes resulting from infection affect the intrarenal blood vessels which matters, rather than the degree of infection. The possibility of the existence of disease in the opposite kidney to account for hypertension existing in cases of unilateral renal stone has been suggested. Against this, however, is the fact that in many cases no evidence of disease in the opposite kidney could be found by the clinical methods available. Furthermore, the incidence of hypertension in cases of bilateral renal stone was no greater than in those of unilateral stone.

HYDRONEPHROSIS

Hypertension was found in only fifty-one, or 13.7 per cent, of 372 patients operated upon for hydronephrosis. Hypertension was observed in only 7.7 per cent of the patients less than fifty years of age who had hydronephrosis. However, when the blood pressure of a patient who has hypertension and who is less than fifty years of age is definitely elevated, it becomes a matter of clinical significance and must be given careful consideration. It is of interest that the degree of pyelectasis apparently was not a factor, since there was no difference in the incidence of hypertension among cases in which pyelectasis was graded 1 or 2 and those in which it was graded 4 (on the basis of 1 to 4, in which 1 designates the mildest and 4 the most severe condition).

RENAL TUBERCULOSIS

The low incidence of hypertension among patients who have renal tuberculosis is noteworthy. Elevated blood pressure was noted in but twelve (7.6 per cent) of 158 cases of renal tuberculosis. It is evident that the pathologic changes in the renal parenchyma resulting from tuberculosis are not conducive to the secretion of a pressor substance. Hypertension might be expected to occur as a result of the extensive atrophic changes which take place in the tuberculous kidney that becomes nonfunctioning as the result of occlusion or caseation. However, in a group of thirty-seven such cases hypertension occurred in only five, or 13.5 per cent. There was no drop in blood pressure among the patients who had a nonfunctioning tuberculous kidney and who were subjected to nephrectomy.

POSTOPERATIVE HYPERTENSION

One of the most interesting results of our study of cases of hypertension in the presence of a surgical renal lesion has been the disclosure of a group of cases in which hypertension developed after renal operation. In a group of fourteen patients the blood pressure was normal prior to conservative renal operation but hypertension developed over a variable period afterward. Postoperative examination in these cases showed evidence of a reduced renal func-

tion with infection in the kidney subjected to previous operation, while the opposite kidney was found to be normal. After removal of the affected kidney the blood pressure became normal in all of the cases studied. Examination of the removed kidney usually disclosed evidence of widespread destruction of renal tissues, which apparently had resulted from operative trauma and secondary infection. The operations which had been performed previously were as follows: nephrolithotomy, plastic operations for hydronephrosis, nephrostomy for renal drainage, ureterolithotomy, nephropexy, repair of ureterovaginal fistula after hysterectomy, repair of persistent vesicovaginal fistula and sigmoidal implantation of ureters. A postoperative increase of blood pressure apparently occurs more frequently after conservative operation for renal and ureteral lithiasis than after operation for other lesions.

RENAL INJURY

Hypertension was noted in only two of thirty cases observed at varying lengths of time after severe renal injury. It might be surmised that cicatricial changes following renal injury might eventually cause hypertension, but a review of such cases at the clinic shows that no change in blood pressure is noted in most cases. It may be inferred, therefore, that in most cases of renal injury either the kidney becomes functionless or the injury is not followed by tissue alterations which cause hypertension.

COURSE OF BLOOD PRESSURE AFTER OPERATION

The postoperative course of the blood pressure was followed in 372 cases in which surgical operation was performed on the kidney. All these patients were traced for at least six months and the majority of them for more than a year. Several patients were traced for as long as five years. Preoperative hypertension had been observed in 198 of these cases and the preoperative blood pressure had been normal in 174 cases. Hypertension was permanently relieved by surgical operation for various renal lesions in sixty-five, or approximately a third, of the 198 cases. Hypertension in the presence of surgical lesions of the kidney was relieved more often by nephrectomy than by conservative operation.

In seventeen cases there was a postoperative drop of blood pressure but the hypertension returned within a few weeks or months after operation. A temporary drop of blood pressure, occurring immediately after operation, often is caused by rest in bed and removal of a toxic irritant. In several cases the blood pressure remained normal as long as two years after operation and then returned to the preoperative level. In such cases it may be inferred that primary hypertension existed and had been temporarily increased by a surgical renal lesion. In order to determine the permanent result of the operation, it is necessary to follow the patient's blood pressure for at least two years.

In the group of patients having hypertension carefully selected for renal operation the number of cases in which the blood pressure will be restored to normal and remain so for a year or more after operation is, unfortunately, very small. Even so, one patient whose blood pressure is restored to normal by renal operation would justify a vast amount of clinical search and investigation. It is difficult to find a group of patients who are more appreciative of the benefits they have received than the patients who apparently were cured by this procedure.

SUMMARY AND CONCLUSIONS

1. A unilateral, non-nephritic or "surgical" lesion in the kidney is not a frequent cause of hypertension. A review of our records shows that the incidence of such lesions among patients having hypertension that are amenable to operation is less than 1 per cent.

2. Surgical removal of a unilateral renal lesion when present will often relieve hypertension.

3. Urographic evidence of deformity in the urinary tract does not always signify that the renal lesion is an etiologic factor of hypertension. Many renal deformities observed in the urogram do not have any clinical significance. Further urologic examination is necessary to exact interpretation.

4. Factors often are present which contraindicate operation in cases of unilateral renal lesions.

5. No specific type of renal pathologic change is observed with hypertension. The essential factor apparently is an intrarenal vascular imbalance which permits the secretion of pressor substances.

6. The renal lesion amenable to surgical treatment which occurs most often in association with hypertension is chronic unilateral pyelonephritis in its diffuse, atrophic or postoperative forms.

7. Acute cortical renal infection or perinephritic abscess seldom is a factor in causing hypertension.

8. The presence of renal stone or hydronephrosis will not affect blood pressure unless there is secondary pathologic change of renal tissues causing intrarenal vascular imbalance.

9. The rôle of secondary infection is important, since hypertension occurred in many cases in which such infection was manifest. However, the deciding factor apparently is not the degree of infection but the consequent lesions in the renal parenchyma which apparently cause intrarenal secretion of pressor substances.

10. The degree of pyelectasis and back pressure did not seem to be a factor, since the incidence of hypertension was no greater in cases in which pyelectasis was of grade 4 than in cases in which it was of grade 1.

11. Hypertension was observed less frequently in the presence of renal tuberculosis than as an accompaniment of other forms of renal disorders.

12. Bilateral renal involvement, such as occurred in many cases of renal stone, hydronephrosis and renal tuberculosis, was not an etiologic factor in hypertension.

13. Hypertension may develop after a previous conservative renal operation. In many of these cases the blood pressure becomes normal after removal of the affected kidney. In every case of hypertension in which there is a history of previous operation, the possibility of etiologic postoperative renal pathologic changes must be considered.

14. Reduction of blood pressure may exist as long as a year or more after operation and yet hypertension may return. In order to determine whether recovery after operation is permanent, it is necessary to determine the patient's blood pressure for more than a year.

15. A follow-up study of cases in which hypertension was present and in which surgical operation was performed showed that in a third of the cases the blood pressure became normal after operation and remained so for a year or more.

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INFECTIONS OF THE URINARY TRACT

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THE urinary tract may be involved by most of the bacteria that affect men. There are certain ones, however, which seem to have a definite affinity for the urinary tract, being isolated with monotonous regularity.

The percentage tables of various investigators vary as to the relative frequency with which these organisms have involved the urinary tract. This is due to several factors:

1. *Methods Used in Taking the Culture.* In spite of the volumes that have been written to the contrary, cultures are occasionally made from voided specimens. In the male, with proper cleansing of the glans penis and collection of the mid-voiding in a sterile container, it is permissible, but still open to error. In the female, the most satisfactory specimen is one which is collected in a sterile container through a catheter which is sterilized by autoclaving. (The lumina of rubber catheters do not become sterile by conventional boiling.) Catheters sterilized by prolonged soaking in germicides frequently retain sufficient germicides to inhibit bacterial growth.

2. *Methods and Care Used in Subculturing.* The vigor and rate of growth of bacteria vary; and like the seeds of many varieties of plants, when the ground is overseeded, the rank growth of the more vigorous plants inhibits the growth of the weaker. Unless a definite effort is made, mixed infections may be reported as a pure growth of the dominant organism.

3. *Sex and Ages of Patients Involved.* Statistics of the special organisms involved in urinary tract infection frequently reflect only the organisms found in certain classes of patients and so fail to record a satisfactory cross section of society. The statistics of one whose practice deals with large numbers of women and children may show an overwhelming percentage of urinary infections of the colon group. One who deals with lusty young males will see many Gram-negative organisms first discovered by Neisser; while those dealing with adults of both sexes with definite foci of infection, will see a larger percentage of Gram-positive cocci in pure culture or combined with Colon bacilli.

The class of patients from which the statistics are made will influence them unduly. For instance, these adult males who reach the urological wards of the general hospitals as the result of urinary retention from an obstructing prostate have usually been examined, manipulated, catheterized and traumatized repeatedly. Their urine, as a result, will present mute evidence of this trauma by the presence of Streptococcus fecalis, Bacillus proteus ammoniae, and Bacillus alcaligenes.

Bacteria occasionally are encountered in the urinary tract as a residuum of a systemic disease: typhoid, para A and B, the dysenteria group of bacilli, the Gram-positive bacilli of diphtheria and the Bacilli welchii.

The micrococcus is always present in the male urethra and is regarded as a saprophyte. Goldstein believes that it is incapable of producing renal lesions and Cook is of the opinion that it rarely involves the bladder. It is not infrequently the only demonstrable pathogen in mild urethritis of young males.

Approximately 80 per cent of the infections of the urinary tract are of bacillary origin and the remaining 20 per cent are due to the coccic type of organism. Bacillary infections occur several times more frequently than do coccal infections

in the urinary tract of females. Coccal infections occur with equal frequency in both males and females.

IDENTIFICATION OF ORGANISMS

When it has been decided that infection is present in the urinary tract, the urine should be centrifuged and a Gram stain made on a smear of the urinary sediment. (Of practical interest is the fact that if the urine is slightly acid, a better stain will be achieved.) This will differentiate the bacilli and cocci, and ascertain as to whether they are Gram-positive or Gram-negative. Gram-positive cocci include the Streptococcus, Staphylococcus, Micrococcus and the Streptococcus fecalis. The common Gram-negative invaders, in general order of frequency, are Escherichia coli, Aerobacter aerogenes, Pseudomonas, Salmonella, Proteus, Shigella and Alcaligenes. It is impossible to distinguish between these organisms by Gram stain. The usual media for culturing colon bacilli is an eosin methylene blue agar plate. On this media, escherichia grows very readily, producing a metallic luster; the aerobacter aerogenes produces large colonies of a pale blue tint, and the proteus produces a spreading growth with a pungent odor.

The Gram-positive cocci can be frequently identified by smear alone. The most common invader is the Streptococcus fecalis which is elliptical in shape and grows in chains. The bunch-grape massing of the staphyloccus and geometrical arrangement of the micrococcus are characteristic. The streptococcus is usually found singly or in short chains. To identify the cocci by cultural methods requires more extensive equipment, the most satisfactory media being brain broth and blood agar.

THEORIES ON METHODS OF INVASION

The possible routes of invasion are as follows: lymphogenous, urogenous, hematogenous, a combination thereof, or direct extension.

For many years, we have accepted the explanation that most urinary infections are due to an invasion of the urinary tract by the colon bacillus, through the lymphatics, the source being the colon. The facts seem to contradict this as it is hard to correlate the theory with certain known experimental data. With the breaking down of the colon group into its various strains, it has been noted that a majority of infected urines show the aerobacter aerogenes strain while cultures of the feces show the Escherichia coli strain.

All experimental work attempting to prove a lymphatic pathway between the colon and the urinary tract, has failed. The normal lymph flow is away from the kidney and the colon through groups of nodes which empty into the thoracic duct. India ink injection experiments of McKenzie proved the lymph flow from the urinary tract to be segmental and non-intercommunicating. The lymph flow is directed to the sacral, inferior vesical, superior vesical, internal iliac, and renal groups of glands. McKenzie's conclusions are worth quoting:

"The most striking feature was that the appearance of the ureter suggested that no dye passed up either the lymphatics of the ureteral or of the periureteral network, no matter the site of the original injection. Whether the dye was placed around the intramural portion of the ureter or at the bladder neck was of no significance. . . . That dye injected in small quantities in rabbits to different areas of the bladder wall, different levels of lower ureteral wall, and into the cervix is absorbed and passes to the common iliac group of glands either directly or through interposed small nodes. The dye then passes upwards toward the thoracic duct and the implication from one series was that the dye then passes to the blood stream and to the kidneys."

The principal interest in discussions of bacterial invasion of the urinary tract centers about the various ways bacteria involve the kidney. This interest reflects the recognized fact that infections of other parts of the tract may be painful or dis-

tressing, but with rare exception, not fraught with the dangers accompanying renal infection.

The more one reads of the experimental work on the methods of invasion of bacteria and their extension to various levels of the urinary tract, the more one is driven to the conclusion that the lymphatics play a secondary part and the blood stream a primary one. McKenzie has shown that bacteria from the normal and abnormal bowel pass into the mesenteric lymph glands (Ravenel, Calmetti, Griffith, David and McGree) and thence into the blood stream.

The descent of bacteria from kidney to ureter and bladder by urine flow is obvious. How bacteria ascend from bladder to kidney is not so obvious.

Ascending urogenous infection has been considered a clinical entity for many years. The experimental evidence to support this has been accepted with reservations. The work of Helmholz seems to be conclusive in proving that bacteria injected into the bladder of rabbits involves the kidney pelvis via the lumen of the ureter. The method of extension into the kidney parenchyma from the pelvis is not so clear cut, and again strong suspicion enters that the blood stream may play a dominant rôle.

In support of Helmholz's histological observations is the work of Graves and Davidoff who, also working with rabbits, showed that regurgitation of vesical contents into the ureter occurs frequently. The ease with thich it occurs, depends upon several factors. I quote:

"Chief among these was good bladder tone which seemed to be indispensable. We learned, for example, that those bladders which were relatively empty and contracted at the beginning of the preparation, produced reflux twice as frequently as those which had been recently distended with large amounts of urine. Moreover, we were soon able to predict the outcome of the experiment by observing whether the bladder wall rounded itself in active resistance to distension, or was becoming filled passively like a toneless sac. Active pressure usually caused regurgitations, passive pressure, never."

How can this be translated into the understanding of certain infections in humans? Ureteral reflux is a condition that would seem to occur more frequently in infants than adults. In doing cystograms on children, reflux of the media resulting in a cysto-ureteropyelogram is common, although its frequency and significance seem to be moot questions. Campbell reports finding it in only 12 per cent of cystograms done on children and considers it evidence of abnormality. It is possible that reflux accounts for the frequency of ascending infection in infants. The preponderance of infections in female infants over males would seem to indicate that the female bladder more frequently receives pathogens from the outside through the short urethra.

Another pathological condition in humans meeting the experimental requirements for ureteral reflux as outlined by Graves and Davidoff, is the urinary retention due to bladder neck or urethral obstruction in adult males. Ureteral reflux, as indicated by cystograms in this group, occurs only when chronic infection in the bladder has stiffened or destroyed the valve-like action of the bladder and intramural portion of the ureter.

Bladder. The bladder may receive bacteria through the ureter from the kidney or by reflux from the urethra or prostate. Prostatic secretion, when abundant, must pass back through the internal sphincter into the bladder. Infection may also spread to the urinary tract from the prostate by way of the blood stream.

The rôle acute inflammations of the bladder play in the production of renal infection by way of the blood stream, is problematical. The work of Macht et al. demonstrating that the bladder exhibits practically no absorptive power, even when damaged by corrosive chemicals, suggests that the bladder does not play an important part in disseminating infection, either by way of the lymph or blood stream.

The low absorptive power of the bladder is in sharp contrast to the absorptive power of the posterior urethra. The ease with which the posterior urethra absorbs is attested to first, by the frequency of general reactions following the instillation of local anesthetics, second, the so-called urethral chill following cystoscopic examination or passage of instruments through the urethra, and third, the occasional chill after voiding through the recently resected prostate. These reactions are due to direct absorption of bacteria or foreign material into the blood stream. W. W. Scott cultured organisms from sixty-two to eighty-two patients who developed fever from urinary infections. Seventy-seven per cent of the infections were bacillary and 23 per cent were coccal. Eighty per cent of the sixty-two had had urethral manipulation and urethral trauma as port of entry.

The most common infection in the upper urinary tract follows the recurring pyuria and bacilluria of the lower urinary tract from infected prostatic secretion when immunity and resistance of the patient is lowered. How this ascent occurs is uncertain, but evidence seems to point to ascending luminal infection of the ureter. Recurring renal infection via the blood stream is of rare occurrence except after urethral trauma.

Infection of the urinary tract by contiguity, is infrequent and study of such cases contributes little to the general knowledge of urinary infections. This infrequency emphasizes how independent an anatomical unit the urinary tract is.

The frequency of involvement of the various portions of the urinary tract increases from above downward. The kidney is the least frequently involved, the urethra most frequently.

It is amazing how much infection can surround the ureter without initiating an urinary infection. The most frequent condition causing pyuria by ureteral involvement from extra-ureteral inflammatory conditions, is appendicitis, with the appendix lying adjacent to the ureter. Tissue from a girl thirty years of age, dying of uremia and hypertension secondary to a long standing osteomyelitis of the hip showed a severe localized periureteritis from the process in the hip, with marked stricture formation and a grade two caliectasis and dilated ureter above. Yet the urine was microscopically negative and there was no history of recurring urinary infection; this, in spite of gradual destruction of the kidney by the septic process in her hip. The bladder wall may frequently be involved secondarily to inflammatory and malignant lesions in neighboring organs to the point of showing leucocytes and red blood cells in the urine, yet there is little evidence to indicate disease of this type results in more than local involvement. When general urinary infection does occur it is by invasion of the kidney via the blood.

Inflammatory involvement may result in the spontaneous discharge of an appendiceal, diverticular or tubo-ovarian abscess into the bladder followed by spontaneous healing or development of a vesico-intestinal fistula.

PREDISPOSING CAUSES TO INFECTION OF THE URINARY TRACT

Clinically, many conditions predispose to urinary infections. Any general consideration must include the factors of immunity and the specificity and virulence of bacteria. The much discussed and little understood immunity factor is important. Any force that upsets the body metabolism lowers resistance and invites bacterial invasion. The most common conditions which disturb resistance are fatigue, chilling and general debilitation. Debilitation may be the result of improper diet, general infection or chronic wasting disease.

Bacteria may often be found in the urine incidental to the bacteremia of a general disease. Since bacteria cannot pass through the kidney without renal damage, actual infection of the kidney must be assumed to have taken place. Such lesions are usually small and parenchymal. They heal

rapidly and only rarely does urinary infection persist after the general infection subsides. Bacteria vary in their degree of virulence and in their specificity. Colon bacilli that are specific in their action on the kidney have been isolated from urinary infections. These organisms, when injected in animals, attack the kidneys and no other organs.

Bacteremia from foci of infection located in teeth, tonsils, mastoid, gallbladder, cervix, prostate and bone, repeatedly expose the kidney to infection. Local factors predisposing to bladder infection are obstruction, trauma and stasis.

Obstruction is defined as interference to the normal outflow of urine. It may be anatomical or physiological and occurs at any level in the urinary tract. Anatomical causes of obstruction to the urinary outflow below the bladder are contracture of the bladder neck, hyperplasia of the prostate, congenital urethral valves, strictures, foreign bodies in the urethra and extreme phimosis. Physiological obstruction to urinary outflow occurs from dysfunction of bladder musculature with spasm of the internal and external sphincters and diminution of the expulsive force secondary to lesions of the spinal cord.

Experimentally, it is impossible to infect the urinary bladder by introducing pathogenic organisms without first producing stasis of urine, tissue damage, or both. This is demonstrated clinically by the procedure of catheterization. It is not feasible to sterilize the male or female urethra in preparation for catheterization. No catheter can be passed through the urethra without carrying bacteria into the bladder. If catheterization is conducted skillfully and with minimum trauma, infections are rare. When bungling and force are substituted for skill, infections are frequent. Painful overdistention of the bladder frequently produces sufficient trauma to the tissues to initiate cystitis regardless of how skillfully catheterization is carried out. The commonest example is the cystitis which frequently follows catheterization of the overdistended bladder after an operation. Trauma to the bladder at the time of surgery may also predispose it to infection, whether introduced by catheterization or by way of the blood stream.

Stasis of urine is a complication of obstruction or trauma. It develops when a bladder decompensates as the direct result of obstruction and may persist after the removal of obstruction. Stasis persisting after removal of urinary obstruction indicates severe damage of the bladder wall as the result of inflammation, diverticulum formation, or neurological dysfunction. Alkaline urine is an excellent culture medium and when kept in the bladder in the form of residual offers ideal conditions for bacterial growth.

In the ureter, the urinary flow depends upon a peristalsis pushing the urine through a lumen of normal caliber. Efficiency may be impaired by excessive narrowing or excessive dilatation of the ureter. Excessive narrowing of the lumen may be due to extrinsic or intrinsic factors. The most common extrinsic factors are aberrant vessels, fibrous bands compressing the ureter, abnormal mobility of the kidney in the presence of a fixed ureter, producing kinking, and pressure from inflammatory masses in nearby organs. The ureter is normally constricted at the ureteropelvic junction, at the crossing of the iliac vessels, at the broad ligament and where it passes through the bladder. Abnormal narrowing at these points exposes them to trauma when small stones are passed, and when ureteritis has been initiated definite stricture may develop and dilatation occur from the obstruction.

Dilatation is most frequently the result of obstruction of the ureteral lumen by a stone. The forceful contractions against such an obstruction and retention of urine above it may bring about a decompensation of the ureter with resulting dilatation. This may be hastened by an intervening infection, with resulting paralysis and destruction of peristaltic activity. Dilatation may occur in the absence of true

obstruction. This was reported by Braasch many years ago. He described it as a destructive ureteritis involving the ureteral wall. The inflammatory reaction so interferes with normal peristalsis that dysfunction and dilatation results. This ureteritis is ascending and progressive, first involving the lower segment of the ureter. Eventually it will involve the entire ureter and pelvis. Dilatation increases dysfunction producing further impairment of drainage which increases dilatation. Extreme dilatation of the ureter may occur without infection. It is of neurological origin and similar to Hirschprung's disease.

Urogenous infections are those that are conveyed from one part of the urinary tract to the other by the urine or from outside the urinary tract by the same media. Clinically, it is to be distinguished from the hematogenous infection by symptoms of lower urinary tract irritability. The urine always shows the evidence of inflammation: pus, blood and bacteria, at the onset. This type of infection is of the ascending variety and is usually due to an organism of the colon bacillus group. This is the largest single group we have to deal with, and is the common type of infection involving females, infants and adults.

This type of infection is characterized by sudden onset with frequency, urgency, tenesmus, hematuria and a fever usually ranging from 100 to 101°F. The patient is conscious of lower abdominal discomfort and the suprapubic area is tender to palpation. With spread of the inflammation, definite tenderness develops upward along the course of the ureters. Extension to the kidneys is then a matter of hours, and is accompanied by chilling, severe back pain, nausea and high fever.

The course of the disease is usually less violent than in hematogenous infections. If the disease is to be self limiting, it becomes quiescent in about ten days. If urinary obstruction exists previous to the infection, or obstruction develops as the result of inflammatory swelling, the disease may be prolonged indefinitely as a subacute infection and produce severe destruction and distortion of the obstructed parts. Even in the absence of anatomical abnormality or neuromuscular dysfunction, there is a marked tendency for the condition to become chronic.

Chronic urogenous infection is the result of previous acute urinary infection which gradually ceased to produce symptoms. This persistence of infection may be due to discontinuing treatment before all evidence of the infection is gone and in making periodic urine examinations for several months as insurance against late recurrence. However, such is not necessarily the case. The initial infection may have been so mild that medical care was not sought. Such infection may manifest itself as a mild, periodic bladder irritability for many years. Such cases are exemplified by the patient who had been in bed a year at home for suspected pulmonary tuberculosis and who, upon being investigated urologically, was found to have a low grade colon bacillus infection of the right kidney with beginning caliectasis and ureterectasis of the upper two-thirds of the ureter. No tuberculosis was ever found and the fever subsided with treatment of her urinary infection.

Pyelitis of pregnancy is most typically an acute urogenous type of infection. Eighty per cent of such infections are bacillary. These patients are also susceptible to streptococci and staphylococci infections of focal hematogenous origin, a susceptibility, however, which does not seem to increase with pregnancy.

I see no reason to hunt far afield for explanations as to the etiology. Several conditions are contributory. Many believe that the bacilli pass from the intestinal tract via the lymphatics and thoracic duct into the blood stream, and thence into the urine with minimal initial damage to the kidneys. Some suggest that the greater moisture in the genital tract during pregnancy increases the possibility of infected materials being transferred from the short urethra into the bladder. A fair percentage

of pregnant women are already carrying pus and bacteria in the urine from a silent chronic infection. Whatever the source of the bacteria, the stasis in the urinary tract during pregnancy provides a fertile field for their growth. In the latter half of the pregnant state, definite anatomical changes take place. A physiological dilatation of the ureters occurs by the sixth month. The histological studies of Hofbauer and of Baird show definite hypertrophy of the wall of the lower third of the ureter. They suggest that this is sufficient to produce anatomical obstruction. On the other hand, Traut, Lane and Tratner have made kymographic readings which show a physiological lowering of the muscle tone of the ureter. This lowering of the expulsive force must affect the bladder as well. Incomplete and difficult emptying of the bladder is not uncommon. The institution of daily catheterization of the puerperal patient until she demonstrates her own ability to empty her bladder satisfactorily has diminished acute pyelitis as a complication of the puerperium.

Clinically, the wide fluctuation of the daily temperature in pyelitis of pregnancy indicates stasis of infected material and impaired urinary drainage. This is diagnostically important, as diffuse lesions of the renal parenchyma give a plateau type of temperature curve with minor daily fluctuations, and emergence of the former into the latter may indicate cortical involvement of the opposite kidney by hematogenous spread.

The history of sudden onset of urinary frequency and bladder discomfort during pregnancy, rapidly followed by chills and fever, back pain, pus and bacteria in the catheterized urine specimen, makes the diagnosis. These are the classical history and findings as encountered most frequently after the fourth month of pregnancy.

The new factors which have been introduced into the treatment of urinary infections in general are especially applicable here. Not many years ago pyelitis was regarded as a troublesome though not too dangerous complication of pregnancy. The most serious result anticipated, unless the infection was of the most fulminating sort, was spontaneous abortion. The patient was kept in bed and treated symptomatically. The urinary antiseptics available were useless in the presence of obstruction, but were administered with varying degrees of faith. If the infection was mild, these measures often sufficed, and were associated with the frequent altering of the patient's posture by the elevation of the head or foot of the bed, until some important observers noticed that such changes made little or no difference in the progress of the disease. But when inlying catheters of sufficient caliber and accurately placed, were inserted in the kidney pelvis, prompt improvement followed. If catheter drainage was not carried out efficiently fever continued and the disease improved only after miscarriage, delivery, or artificial interruption of the pregnancy. The risk of losing the child was high; the patient had a prolonged, severe illness, and was left with a permanently damaged urinary tract. With the realization of the havoc being wrought by procrastination, it became the accepted procedure to institute immediate ureteral catheter drainage following a chill. Catheters of No. 10 to No. 14 French were used. This was, and at times continues to be, our most satisfactory method of controlling infection and renal damage. However, these catheters are not without danger. A marked foreign body reaction occurs in the ureters with a resultant severe periureteritis. Adequate asepsis in handling the catheter is impossible. Staphylococcus, Streptococcus fecalis, and other organisms may be introduced into the kidneys from the outside, resulting in superimposed infection with bacteria much more virulent and destructive than the original organisms. The patient may become intolerant to the catheter or they may not be able to dispense with the catheter even after weeks of catheter drainage. If these conditions develop, nephrostomy or pyelostomy are to be considered.

With the advent of the newer bactericides, for the first time we have been able to sterilize the urinary tract in the presence of urinary obstruction. This was first accomplished by Helmholz with the ketogenic diet in the obstructed uropathies of children. However, the ketogenic diet is not satisfactory in pyelitis of pregnancy because of the difficulties of administration. Mandelic acid was the first drug which would abort early urinary infection of pregnancy and sterilize the urinary tract with any degree of regularity.

Sulfathiazole and other sulfonamide derivatives are even more efficient. With such potent drugs at our disposal, there is a definite tendency to rely exclusively on them, with the result that at times, we wait too long to institute catheter drainage. It is an unfortunate fact that bacteria most resistant to the sulfonamide drugs are the ones which cause the most renal damage.

WHEN SHOULD THE DRUGS AND WHEN SHOULD CATHETER DRAINAGE BE USED?

If the drug is going to be effective, improvement will be noted within forty-eight hours. This improvement is indicated by a downward trend in the fever and pulse, subsiding renal and bladder tenderness, and lessening of the bacterial content of the urine. If no change has taken place within that time, inlying ureteral catheter drainage should be instituted immediately and combined with small doses of the drug as prophylaxis against outside contamination.

If pre-existing renal damage or lowered renal function is known to be present, ureteral catheters should always be used, but if they prove unsatisfactory, surgical intervention is to be seriously considered.

HEMATOGENOUS INFECTIONS OF THE URINARY TRACT

Acute hematogenous infections are caused by bacteria which are brought to the kidney by the blood and produce multiple foci of infection throughout the cortex and medulla. The common infecting organism which we encounter is the Staphylococcus aureus. English observers, however, regard the Staphylococcus albus as the most frequent etiological agent. Streptococci may produce the same type of lesion and as I have previously mentioned, the colon bacillus has at times a specific action on the kidney when injected into the blood stream. Bacilli capable of producing this type of lesion must be rare. The portal of entry is either the mucous membrane or skin. The condition occasionally complicates acute infections such as pharyngitis, furuncles, carbuncles, osteomyelitis, etc. The source may also be such chronic foci of infection as the teeth, the tonsils, the gallbladder, cervix, or prostate. A surgical operation on infected tissues may be the initiating event.

These hematogenous infections may be classified according to their severity into two groups: acute and subacute. In the acute there is a sudden onset with diffuse, upper abdominal pain followed by nausea, vomiting, collapse and coma. Fever, pulse rate and leucocytosis will be high. All the signs of sepsis dominate the picture. The condition is usually associated with a recent pyogenic lesion of the skin, as a boil, carbuncle, or septic wound. These may be healed at the time of onset; and if definite inquiry as to the recent occurrence of one of these lesions is not made or the patient is unable to give a history, this fact may not be elicited. To complicate the diagnosis, the urine shows no pus or blood. There may be the usual trace of albumin which can occur in any febrile illness. The two distinguishing features are: (1) Pain and tenderness in the costomuscular angle. This may be difficult to elicit, but should be present before the diagnosis is made. The habit of using a forceful blow over the renal area in attempting to prove this tenderness is to be condemned. The method is inaccurate. It causes unnecessary pain and seems capable of doing actual harm. (2) The other finding is the regular occurrence of cocci in the centrifuged, stained sediment of the catheterized urine specimen. (Cultures are usually negative.)

As the disease progresses, it may be followed by pain in the renal areas. This may be bilateral though usually it is unilateral. As the infection regresses in one kidney as indicated by disappearance of tenderness, it may invade the opposite kidney and be followed by identical clinical symptoms experienced in the other kidney. The disease is usually self limiting, running a febrile course in five to ten days. As the fever subsides, or if the course is prolonged, pus may appear in the urine, with or without Bacillus coli. This is interpreted as spontaneous drainage of cortical abscesses into the tubular system of the kidney. The Bacillus coli are thought to be secondary invaders.

If the course is prolonged, even though the general level of the fever falls and begins to fluctuate, a carbuncle of the kidney or parenchymal abscess may be developing.

The subacute type may begin insidiously with backache and persisting low-grade fever of 99½ to 101°F., and with a leucocytosis under 15,000. Costomuscular angle tenderness will be elicited and cocci demonstrated in the Gram stain of the urine. If the course is protracted, a search for a focus of infection should be made.

PERINEPHRITIC ABSCESS

Perinephritic abscess is a collection of pus surrounding the kidney. It is a complication of renal cortical inflammation. The extensive perinephritis that exists in these infections usually develops as an extension of a cortical abscess into the perinephritic fat. The rapid healing power of the kidney often results in healing of the renal cortex, while the low immunity of the perinephritic fat allows for continual development of the abscess as an independent process.

The early signs and symptoms are identical with carbuncle of the kidney or of a small cortical abscess. As the process becomes older and more localized, the fever recedes and the daily fluctuation may not exceed one to two degrees. Night sweats are frequent and flank pain, tenderness and leucocytosis are the rule. Regular examination of the painful area will reveal a mass of increasing size and a progressive increase of the rigidity involving the muscles of the upper abdomen of the involved side. Difficulty in walking and painful extension of the thigh indicates extension of the abscess into the iliopsoas muscle. The kidney, in advanced perinephritis, has usually recovered its function, and excretes normal uninfected urine. The *x*-ray findings are definite. The plain negative may show fuzzing and an increase in size of the renal shadow, loss of the iliopsoas shadow, loss of normal renal mobility with respiration and elevation of the diaphragm on the affected side. Pyelo-ureterograms are negative or show displacement and pressure deformity from an extrarenal mass. The treatment is surgical drainage. Many abscesses are multilocular and thorough perirenal exploration is necessary at the time of surgery.

In the acute infections, three questions immediately arise: Is there pus in the urine? What type of bacteria are present? Is there any obstruction to urinary outflow? The first two questions are answered by microscopic examination and Gram stain of the specimen of urine obtained through a catheter and centrifuged at high speed. The catheter will also determine whether there is bladder neck obstruction with residual urine in the bladder. The question of urinary tract obstruction above the bladder can be determined either by cystoscopy or intravenous pyelography. If there is pus in the urine or if there is presumptive evidence of urinary obstruction, a plain *x*-ray of the kidneys, ureters and bladder, followed by cystoscopic examination, is the procedure of choice. If the urine is free of pus and urinary obstruction is not anticipated, intravenous pyelography often gives all the desired information.

When cystoscopy is employed, it should be accomplished as rapidly and with as little disturbance to the patient as possible. Preliminary injection of indigo carmine is

helpful. Its prompt appearance in good concentration indicates that obstruction, if present, is not complete and the inflammatory process has not involved the kidney sufficiently to cause it to cease functioning. I have never observed an acutely inflamed kidney that demanded surgical removal as long as it was able to excrete dye or the organic iodides.

MANAGEMENT

In this day of specific therapy of infections, the importance of intelligent attention to the general management of urinary infection is often neglected. It is well to remember that these conditions were successfully treated from the patient's standpoint long before we had our present drugs. The continued use of alkaline therapy is largely based on the relief it affords the patient of nausea and bladder irritability and not for any specific effect on the bacteria of which it possesses none. Immediate attempts should be made to relieve the patient of his pain. Opium, or its relaxing alkaloids papaverine and pantopon, take preference over morphine or codeine. The azo dyes are useful in allaying bladder pain by their anesthetic action on the bladder mucosa. Their action as a bacteriacide seems neglible. Bladder lavage with normal saline or isotonic boric solution, followed by the instillation of one-half ounce of metycaine or the colloidal silver salts, will give relief and allow time for the action of the opium derivatives. An intramuscular injection of an ampoule of calcium gluconate or 5 cc. of deproteinated insulin free pancreatic extract is helpful.

Heat in any form gives relief from discomfort. The local application of warm towels to the perineum, sitz baths, and infra-red lights can be used. Short wave diathermy applied with external electrodes or with combined external and vaginal or rectal electrode is especially effective. Considerable importance should be attached to the control of bladder spasms. The satisfactory control of these spasms is necessary in localizing bladder infection and in preventing renal involvement.

Except as an adjunct to specific therapy, in the acutely ill, special diets are of no value. Small frequent feedings are more successful than three daily meals. See that favorite articles of food that the patient likes (not the physician) are always available and are placed before him when his fever is receding or between febrile reactions; unless a previously known sensitivity for spices or condiments exists, do not omit them; keep the food as appetizing as possible. Fluids are of great value. They combat toxemia by keeping up the tissue fluids and promote drainage by mechanically irrigating the urinary tract. A chart of the fluid intake and urinary output should be kept and the average daily output should be between 2,000 and 3,000 cc. of fluids; this may require an intake of 4,000 to 5,000 cc. This amount is too much to ask the patient to drink. It is less burdensome and more effective when administered intravenously. Fluids are best administered intravenously in isotonic concentrations. This is more important in the aged than the young. If concentrated solutions are used, 5 per cent glucose in normal saline is the solution of choice. Any danger of overloading the circulation with intravenous fluids can be avoided if the lung bases are checked for moisture before each administration. Excess chlorides and tissue overloading may be avoided by watching for edema. Pressure on the skin of the forehead will leave an impression of the whorls of the thumb pads in early edema. If this occurs, lower the fluid intake and use solutions without sodium chloride. Multiple small transfusions help to combat toxicity and should be used to combat progressive anemia in the hemolytic infections.

All patients with infections of the urinary tract should have sufficient urological investigation to determine the types of infecting organisms, the parts involved, and the presence or absence of obstructions to the urinary outflow. The proper time to make the investigation and the thorough-

ness of it depends upon the stage of the illness and the clinical judgment of the physician.

When an ureteral catheter is passed up the affected side and an impassable obstruction is encountered or definite retention in the pelvis of the kidney is noted, adequate drainage above the obstruction must be established and immediate surgical exploration may be indicated if the obstruction is complete. When an inlying catheter is placed, it should be at least No. 8 or a No. 10 French, and should be adjusted by the *x*-ray for most efficient drainage. When stricture or stone blocks the passage of a catheter, immediate surgical drainage by nephrostomy or pyelostomy is indicated. When there is no retention of urine in the kidney pelvis, I cannot too strongly condemn the routine placing of inlying catheters. No purpose is served and the urinary tract is thrown open to the trauma of a foreign body and the possibility of receiving infecting organisms more virulent than those of the existing infection.

Active etiological foci of infection are not common in the acute infections. They are much more common in the recurrent subacute and chronic infections. They should be diligently sought for and eradicated when present.

The acute parenchymatous infections at times require nephrectomy as a life saving measure. Increasing conservatism has been exercised in this condition with equally good results. Since the advent of the sulfonamides, the indications for surgical removal of the infected kidney are increasingly rare. However, a persisting unilateral infection as indicated by constant unilateral costomuscular angle tenderness, increasing lethargy and toxicity and a steadily rising pulse to 100 or above, which fails to fall with the slight recesses in fever, is an absolute indication for nephrectomy.

Fever and unilateral tenderness in this type of infection lasting over ten days, is strong evidence of cortical necrosis or perinephritic abscess, and indicates surgical exploration. However, one must be certain that the continued fever is not caused by a secondary cortical infection developing in the opposite kidney.

CHEMICOTHERAPY

The past decade has been marked by a great advance in the development of drugs and understanding of the principles by which older drugs are more effective and by which certain conditions may be produced in the urinary tract to inhibit and destroy bacteria. Of added importance has been the detailed bacterial studies of infected urines, the isolation of the various types and strains of bacteria to varying degrees of acidity, alkalinity and concentrations of proposed bactericides.

The most effective bactericides in common usage today are divided into two main groups: those which depend upon acidification of the urine for their action, and the sulfonamide compounds.

It has been known for years that increased acidity of the urine inhibits bacterial growth. The importance of this has been emphasized in recent years. Acid urine with a hydrogen ion concentration of 5 or below inhibits many of the common bacteria found in urinary infections and indeed, is at times bactericidal for some strains of the colon bacillus. One of the most efficient urine acidifiers is ammonium chloride, preferably given in enteric coated tablets. Four Gm. a day is an average dose that is usually well tolerated. It may be increased to 6 Gm. a day without producing too much gastric irritation and if necessary this dosage may be continued for several weeks. The use of this drug alone will often give clinical relief of burning and frequency of urination within eight to twelve hours. It is much more effective if combined with mandelic acid or methenamine.

Methenamine is the only one of the older drugs that is effective with any degree of regularity. Its greatest drawback is the frequency with which the formaldehyde produces bladder pain and irritability before bacteriostasis can be produced. To

be effective a daily dosage of 3 to 6 Gm. must be used and the urine must be kept at a hydrogen ion concentration of 5 or below.

Mandelic acid is our most efficient urinary antiseptic outside of the group of sulfonamide compounds. It is an organic acid similar in action to beta-oxybutyric acid which is produced by the ketogenic diet. It was introduced by Rosenheim as a substitute to overcome the technical difficulties of administering the ketogenic diet. It is excreted unchanged by the kidneys into the urine. At a urinary hydrogen ion concentration of 5 to 5½ and a concentration of ½ to 1 per cent, it is bactericidal for all the Gram-negative bacilli found in the urinary tract. It is equally effective for Streptococcus fecalis and Staphylococcus aureus. Clinically, it is less effective in proteus ammonia infections, because of the difficulty in obtaining the proper urinary acidity.

One of the most potent reasons for the dissatisfaction with the drug is gastric irritation, nausea and vomiting. This is frequently caused by using the more irritating ammonium salt of mandelic acid. The combined enteric coated tablets of mandelic acid and ammonium chloride are less irritating, but have the inherent disadvantage of a combination tablet. The dosage rates of the two drugs cannot be varied and to get the proper therapeutic amount, the patient has to take twenty-four or more of the tablets daily.

Calcium mendelate has the advantage of producing an acid urine as well as one which contains the mandelic acid but it may be necessary to reinforce its acidifying properties with ammonium chloride.

The most satisfactory results have been attained with the sodium salt of the drug, given in a 10 per cent solution and the acidity of the urine controlled by the separate administration of 0.5 Gm. enteric coated ammonium chloride tablets. This is admittedly more cumbersome than a streamlined tablet containing both drugs but it has definite advantages. Proper acidification is easily obtained, adequate amounts of mandelic acid are injested, and gastric irritation occurs less frequently from the sodium salt than the calcium and ammonium salt.

A prescription suggested by Clark some years ago follows:

Mandelic acid	48 Gm.
Na HCO (3)	25.6
Lemon syrup	90 cc.
Aqua q.s.ad	480 cc.

Sig. 2 tbsp. after meals and at 8 P.M.

This should be combined with 8 to 16 of the 0.5 Gm. enteric-coated ammonium chloride tablets daily. They are best tolerated before meals.

This treatment is of no value in the presence of renal damage, because the damaged kidney is unable to excrete the drug in sufficient concentration to produce bacteriostasis. It must be used with great care in Bright's disease as it may further depress renal function, elevate the blood urea and increase hypertension. Coarsely granular casts may occasionally be noted in the urine during administration. This is a sign of renal intoxication and is an indication to lower the dosage or withdraw the drugs.

It is not a drug to be taken in small amounts over a long period of time. It should be given in full doses in definite courses not to exceed ten days, followed by a period of rest and then by another course if necessary. If two trials fail to sterilize the urine, further attempts will be unsuccessful.

When this drug is being used, the patient should be placed on an acid ash type of diet and fluids limited to about 1,000 cc. daily. Acidification therapy with administration of mandelic acid is especially valuable in the acute nonspecific infections of the lower urinary tract with or without lower urinary retention. Complete symptomatic relief is frequently a matter of hours and sterile, pus free urine, is obtained in several days.

Five years ago, sulfanilamide was added to our armamentarium. Since then, a series of the sulfonamide type of drugs have

followed in rapid succession. The present favorite is sulfathiazole, which seems to be the most effective clinically. These drugs are especially adaptable to the treatment of urinary tract infections because the drug can be excreted in therapeutic quantities by kidneys too damaged to get any effect from the use of mandelic acid and acidifying drugs and incredibly small doses will exert a bacteriastatic action on the urine. These clinical observations have been emphasized experimentally by Helmholz, Cook and others. They have definitely shown that doses as low as 5 to 10 gr. daily are clinically effective in all of the ordinary urinary infections with the exceptions of the Pseudomonas and Streptococcus fecalis. The Streptococcus fecalis is extremely resistant unless the urine is strongly acidified to a hydrogen ion concentration of 5 and below before using sulfathiazole. Of great importance is the effectiveness of the drug in alkaline incrusting infections. For the first time, we are able to save these people from constant misery and progressive renal destruction. When giving small doses, the urinary output must not exceed 1,000 to 1,200 cc. of urine daily.

In acute hematogenous infections, dosages must be sufficient to hold the blood concentration of the drug at 2 to 5 mg. per cent.

The remarkable specificity of the drug is such that if clinical improvement is not noted within three days, the drug is valueless and should be withdrawn. Extensive renal damage, obstruction, and foreign bodies diminish the effectiveness of these drugs as bacteriostatics, just as in the past this has been true of the older less efficient urinary antiseptics.

Intolerance to sulfathiazole should be watched for at all times. If any doubt exists, the drug should be withdrawn. Toxic reactions bear no relation to the amount of drug being administered. They occur just as frequently on 7½ gr. a day as on 60 gr. daily. These reactions may be puzzling. A good rule is to withdraw the drug from any patient who develops chills and fever several days after starting sulfathiazole, if no renal tenderness can be demonstrated and the white blood count is 10,000 or below. Photophobia and conjunctivitis may precede the fever and the more common signs, nausea, vomiting, and skin rashes, are familiar to all. Any of these reactions indicate immediate withdrawal of the drug.

The effectiveness of sulfathiazole and related compounds has required us to change our conception completely of the management of urinary infections. The haphazard administration of the drugs cannot be too strongly condemned. However, they will accomplish what was impossible a few years ago, a sterile urine in the presence of severe upper and lower urinary tract obstruction and bacteriostasis and symptomatic improvement in the presence of foreign bodies, stones and incrusting tumors.

The relief of symptoms may lull both clinician and patient into delaying adequate investigation until irreparable damage has taken place as a patient may feel better to have the secondary infection in his tuberculous bladder controlled but his renal tuberculosis may continue unchecked; or the bleeding and incrustations of bladder tumor may be temporarily improved, but the cancer has not been affected. With these new, more effective weapons at our disposal, there never has been a time when the duty of those who assume the responsibility to treat infections of the urinary tract has been clearer.

They must be fully cognizant of the possible paths of invasion and the predisposing factors which precipitate invasion of the urinary tract. They must be able to recognize physiological variation as well as anatomical abnormality in the urinary tract, and they must treat the patient as an individual, if success is to crown their efforts; they must know intimately the details of the problem to be dealt with and the limitations and dangers of the drugs employed.

Someone has said that the reason there are so many chronic urinary infections is because acute infections are so poorly treated. It is not enough to carry the patient past his febrile period to asymptomatic relief, but treatment should be continued until cure has been obtained or all possibilities of cure have been exhausted. If cure results, periodic examinations should be made over an extended period of time to insure its permanence and mitigate against late recurrence. Then and then only, have you discharged your responsibility to your patient.

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THE DIAGNOSIS OF RENAL TUBERCULOSIS

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MOST urologists are of the opinion that renal tuberculosis is seen less frequently today than in the past two decades. This is due to the reduction in the incidence of pulmonary tuberculosis, as shown in recent data published by the National Tuberculosis Association, the control of the bovine form of infection and the more common use of pasteurized milk. Renal tuberculosis is, I think, more commonly encountered in the larger metropolitan centers and in the Northern and North Central states than in the Southern and Southwestern states.

HISTORICAL

Since it is only in quite recent times that means have been available for the early recognition of renal tuberculosis, it was inevitable that indications for its treatment should have lagged behind the general march of progress in the field of medicine. And because in its early stages clinical symptoms may be nil, or may differ in no way from those of other common urinary affections, for a long time only advanced cases of renal tuberculosis were recognized, in which the bladder, ureters and kidneys had all begun to show extensive destruction, such as were seen in certain museum specimens. It was not strange that the infection was universally believed to be of an ascending nature, starting in the bladder, where the first symptoms were observed, and supposedly reaching the kidneys little by little over a long period of time. Such changes, moreover, were so complex that the idea of their eradication by nephrectomy was inconceivable.

The French urologist, Tuffier, wrote in 1892: "Surgical intervention in renal tuberculosis should be limited to palliative measures. Retention of pus or the presence of intense pain are the principal grounds for operative measures." The idea of nephrotomy was accordingly taken up and the procedure carried out in certain severe cases, but it resulted at best in mediocre relief, and at worst in disaster. Albarran was quick to call attention to the persistence of infection and intoxication and to the invariable failure of the fistula to close. Cachexia increased and none of the nephrotomized patients survived more than one and one-half to two years.

All of this was revolutionized with the discovery that the tubercle bacillus primarily invaded the kidney from some other tuberculous focus within the body and had been secondarily passed on to the bladder. Actually, through an error of diagnosis, Peters in 1872 had done the first tuberculous nephrectomy. But it was not until between 1892 and 1900 that nephrectomy was generally employed in renal tuberculosis and at first the results were far from satisfactory, owing to the difficulty of determining the status of the opposite kidney. In 1897, Albarran published an article in which he set forth the merits of the new surgical method, which, with his introduction of ureteral catheterization as a means for the determination of the differential function of the two kidneys, placed the treatment of the tuberculous kidney on an entirely new footing. He showed the advantages and the necessity of early intervention and carried out nephrectomy in cases in which the lesions were so slight as to seem almost latent. The new method was taken up with enthusiasm by the leading surgeons of that time throughout Europe, and in the United States by such men as Kelly, Meyer, Bevan and others. Today early nephrectomy is universally accepted as the only curative surgical treatment in cases in which the opposite kidney is sound.

In the years that have intervened since the introduction of ureteral catheterization, means of diagnosing renal tuberculosis have been multiplied and today it is usually possible to make an early diagnosis on the basis of the history, the clinical findings, the results of cystoscopy, urography, and the finding of the tubercle bacillus in the urine. With an early diagnosis renal tuberculosis has passed into the category of curable diseases.

ETIOLOGY

It is now fully established that renal tuberculosis is definitely secondary to some primary focus of tuberculous infection elsewhere in the body. This focus may be a small gland in the mediastinum or mesentery, an incipient apical tuberculosis, a bone lesion too slight to attract attention, or associated with a lesion of the prostate, seminal vesicles or epididymes.

The incidence of associated genital lesions is high. Caulk found co-existing lesions of the prostate, seminal vesicles and epididymes in 75 per cent of his cases of renal tuberculosis. Young found tuberculous epididymitis associated with 26 per cent of his patients suffering with renal tuberculosis and Bumpus and Thompson in studying 606 cases of genital tuberculosis, found 42 per cent associated with renal lesions.

The infection of the kidney is hematogenous in origin in the vast majority of the cases; however, the possibility of a lymphatic invasion in some cases cannot be denied. Thus Hess cites a case in which infection was demonstrably passed by the lymphatic route from a tuberculous focus in the cecum to the right kidney, there being, as is well known, direct communication between the lymphatics of these organs. A third route taken by tuberculous infection is from one kidney to the other by way of the bladder through vesico-ureteral reflux. When the region of the trigone has become infected, changes in the ureteral orifices make it possible for the bacilli that have traveled down one ureter and into the bladder to enter the opposite ureteral orifice readily and ascend to the kidney.

INCIDENCE

Sellaras and von der Becke found in a study of 1,724 patients with urological symptoms fully 16 per cent with renal tuberculosis. Kuster reported renal tuberculosis in 10 per cent of patients dying of tuberculosis. Caulk found tuberculosis responsible for 19 per cent of his nephrectomies while Kronlein showed tuberculosis of the kidney to occur in 28.9 per cent of all surgical operations on the kidney. These figures contrast quite markedly with my findings of four cases of renal tuberculosis in 1,131 consecutive urological examinations.

THEORY OF BILATERAL RENAL INVOLVEMENT AT OUTSET

Two principal types of renal tuberculosis exist: (1) A sudden, fulminating, bilateral type, which is part of a general miliary tuberculosis, in which the parenchyma of the kidney is suddenly overwhelmed by multiple emboli of the tubercle bacillus, with death ensuing so swiftly that the diagnosis is usually made at autopsy, and (2) a slow developing, chronic type, which may remain silent for a long time after the blood-borne infection has reached the kidney, and which is amenable to surgical treatment. It is this second type which is under consideration here.

Whether the chronic form of renal tuberculosis in unilateral or bilateral at the outset is still debated among urologists. Hammond stated that it begins in one kidney in 92 per cent of the cases, but that those dying of it have both kidneys involved in 70 per cent of the cases. Hess holds that it has been clinically established that it may remain confined to one kidney for many years. In a study of experimental renal tuberculosis in guinea-pigs, Medlar and Sasano, who observed a striking resemblance of the lesions in these animals to human lesions, found that in 88 per cent of their series the lesions were bilateral.

Cortical lesions were the most common, but medullary lesions were more destructive to kidney tissue. All their guinea-pig lesions were hematogenous. According to Caulk, both kidneys evidently receive an equal share of the tubercle bacilli yet only one becomes diseased at first, while the other, possibly because of local resistance, freedom from allergy, or inherent anatomical integrity, does not share in the infective process until later when bilaterality will almost surely result from infection in the bladder, by ureteral regurgitation of its contents to the sound kidney.

It would appear that the generous blood supply of the kidneys must enable them to overcome a large number of early infections on one or both sides. Chute believed that bilateral infection is frequent, but that this rich vascularity of the renal tissues offers a strong defense which serves them efficiently until some obstructive condition weakens their normal resistance and gives a foothold to the Koch bacillus. Braasch and Sutton reported 2,200 cases of renal tuberculosis observed at the Mayo Clinic during the years 1910 to 1934, and clinical evidence of bilateral involvement was present in 291 cases (13 per cent).

Cabot is of the opinion that the infection is always bilateral at the outset and that the lesion may become surgical in importance in only one kidney in the early course of the disease.

Thomas and his co-workers believe that renal tuberculosis is a local manifestation of a constitutional disease, is practically always hematogenous and may be primarily a bilateral infection. The lesions may be destructive or inflammatory; those that are inflammatory may heal. They believe that healing frequently occurs in initial tuberculous lesions of the kidney; that there is room for the view that the present strains of the tubercle bacilli may be undergoing a change, and that also the resistance of renal tissue to these strains may be undergoing a change, all of which, with improvements in the modern methods of treatment, greatly enhance a favorable prognosis.

SYMPTOMATOLOGY

So slow is the progressive invasion of the kidneys by Koch's bacillus that the infection may have been implanted for years before any symptoms arise. The first symptoms are those of cystitis. The patient complains of frequency, especially at night, and suffers with dysuria and urgency; in children incontinence is to be expected and it is sometimes present in adults. If the trigone is involved, the pain will be especially acute at the close of micturition. In most cases lumbar and renal pain supervene and the kidney may become tender on palpation. Occasionally, profuse hematuria is an early symptom—in some cases the first. The urine may be loaded with pus and red blood cells are present microscopically. Loss of weight and general debility are frequent. But none of these symptoms are pathognomonic of renal tuberculosis and it is obvious that the diagnosis cannot be made on these alone. One point, however, must be stressed, namely, that in all cases of cystitis that persist in spite of treatment, the possibility that the patient is suffering with renal tuberculosis should not be overlooked. Berthon points out that in elderly men the symptoms are so often suggestive of an infected prostatism that this warning is especially important.

A deceptive feature in some of these cases is the occurrence of long remissions free of painful symptoms, sometimes for years and with a relatively clear urine. Bernard and Heitz-Boyer point out that this remission is only clinical; the disease is slowly at work in the dark and that for every cavity that is obliterated a new nodule is forming. According to the patient's general resistance, the progress of the hidden renal tuberculous process will be more or less rapid; and it is now beginning to be recognized by urologists that general constitutional treatment can strongly influence the length of the remissions and hold off the rapidity of the destructive process. Thus Thomas emphasized the rationality of putting a patient to bed and employing a

comprehensive hygienic regimen. He states that repeated instances have been observed in which patients under intensive constitutional treatment for other tuberculous conditions, which at the time precluded any surgical trauma, have experienced marked amelioration of urogenital symptoms. Thus it would appear that there is a constitutional factor behind these alternating remissions and exacerbations of the symptoms of renal tuberculosis; and that the exacerbations, if they cannot be prevented from their inexorable march of destruction, can at least be subject to some control through a better understanding of the factors at work.

URINARY FINDINGS

Urinalysis. The most important evidence of the presence of renal tuberculosis is furnished by examination of the urine. The earliest sign here is usually the finding of pus cells microscopically which may exist in a sparkling clear urine. Red blood cells are frequently found throughout the entire course of the disease. In the later stages the blood cells may be masked by the pus cells, which are never absent in renal tuberculosis and which may exist in enormous numbers, causing marked turbidity.

Casper states that he has found the Koch bacillus in 80 per cent of cases if the correct staining technic was used. It is not easy to find this bacillus, since there are seldom many of them in the amount of urine available for study. Their number varies, often being greater in the early than in the late stages of the disease. It diminishes when renal bleeding occurs, but Israel points out that they are numerous when bladder tuberculosis is far advanced. The bacilli are not, however, discharged into the urine if the tubercle formation has not yet broken through into the renal pelvis, or if for any reason the diseased kidney or its ureter is shut off from the bladder. Rapid changes in the degree of turbidity may occur as cavities break open or become closed in the kidney.

Caulk utters a warning that the presence of tubercle bacilli alone does not warrant nephrectomy, since the number of these organisms bears no relation to the gravity or extent of the renal lesions. Bacilli, plus microscopic blood, plus pus, are necessary to constitute the picture before any drastic action is carried out.

Albuminuria is a rather constant finding, so constant, indeed, that Wildbolz considered its absence almost a negation of the presence of tuberculosis in the kidney.

As early as 1897, Melchior announced that tuberculous urine is always acid in its reaction. This was confirmed by Suter in 1907 and later by Caulk and many others. This acidity has been widely used as an argument to account for the allegedly constant absence of other infectious organisms from the urine. In view of the general belief that had been given to the idea that the urine by its acidity inhibits the growth of other organisms in renal tuberculosis, Alcorn and Buchtel in 1938 undertook to determine whether this was actually true and found that in 38 (44.7 per cent) of the cases of renal tuberculosis studied, other organisms than the tubercle bacillus were actually found, thus refuting the time honored belief that urinary tuberculosis cannot be associated with other organisms. These findings agree with those of Kretschmer and of Sas and Szold.

METHODS OF IDENTIFYING THE TUBERCLE BACILLUS

(*a*) *Stains.* It has been well said by Marion that the difficulty with the tubercle bacillus is not the recognition of it but the discovery of it in a urine in which it may be practically lost in a morass of pus. These bacilli occur very sparingly in urine, and both experience and patience are required to make the necessary studies.

Only by a special staining method can these tubercle bacilli be brought out on the slides. This is accomplished by the Ziehl-Neelsen (carbol-fuchsin) procedure. Under a proper technic the bacillus, if present, is seen standing out clearly and unmistakably

in red clumps against a background of blue pus cells. Before staining, it may be desirable to digest secondary infecting organisms by the antiformin method, but even so, an almost endless series of slides may have to be examined before any of these elusive bacilli are found.

Hyman and Mann, in a study of the urine from 126 cases of renal tuberculosis, found that the bacterial content of the urine varies considerably at different stages of the disease. If a new focus is opening up, the organisms may appear in veritable showers, which no one could miss; whereas in advanced cases with thick purulent urine and numerous secondary infecting agents, it may be impossible to find them even in smears from an extirpated kidney.

The percentage of positive results from studies of smears varies widely in the reports of different workers. Wildbolz, for example, found the bacilli in 90 per cent of 155 nephrectomies, Casper in 80 per cent and Hyman and Mann in 65 per cent of 102 operative cases.

The smegma bacillus, which also stains red with the Ziehl-Neelsen stain has sometimes been mistaken for the tubercle bacillus; but if proper care is taken in collecting the urine specimen, no confusion should result.

(*b*) *Guinea-pig Inoculations.* When smears give no decisive results, there remains the more time consuming method of guinea-pig inoculation with the urinary sediment, introduced intraperitoneally, subcutaneously or in the groin. This procedure entails a waiting time of some six weeks which has seemed to some urologists a needless and regrettable waste of time. To others, perhaps the majority, this delay presents no disadvantages in view of the very slow course of the disease. Attempts to shorten the waiting time by various expedients have not been wholly satisfactory, and the method, as developed in 1882, is being used today with a technic that has undergone little modification. All urologists agree that it is a very valuable aid, but admit it is not infallible. As to the percentage of error, opinions differ considerably. Thus Barney and Jones reported that they found it 99.5 per cent accurate in 197 proved cases of renal tuberculosis at the Massachusetts General Hospital, only two cases giving negative results. They expressed the view that, when the test is carefully carried out, it is by far the most accurate diagnostic aid available, and that a negative test is accordingly of indisputable value.

According to Casper, who values highly this mode of procedure, there are two possible ways to account for negative results in positive cases: (1) In an early stage of renal tuberculosis, there are periods when no tubercle bacilli, or only very few, are present in the urine, which is therefore incapable of infecting the guinea-pigs. This would be the case if the process had not yet reached either the renal pelvis or the canaliculi. (2) The tuberculous kidney is at times shut off and no tuberculous product reaches the urine. Casper never operates on a tuberculous kidney until guinea-pig inoculation has proved that the opposite kidney excretes a negative urine. He insists also upon two consecutive negative results of such inoculation.

Morse and Braasch have not found guinea-pig inoculation sufficiently reliable to be trusted unless confirmed by uroscopy, cystoscopic data, clinical data and examination of the genitals. Renal tuberculosis should never be diagnosed based on inoculation alone. In eight of a series of forty-five cases of proved renal tuberculosis (17.7 per cent) the guinea-pig findings were negative. In 109 cases of proved tuberculosis of one kidney, with the other thought to be a normal mate to a tuberculous kidney, guinea-pig inoculation was positive in eighteen per cent. When the inoculation made with urine taken directly from the kidney is positive and any of the data mentioned are positive, the diagnosis of renal tuberculosis is evidently correct. But such a diagnosis should never be made on the inoculation without other supporting data.

Comparing the relative advantages of smears and guinea-pig inoculations, Hyman and Mann give preference to the smear as a simpler procedure and one that can be rapidly carried out. They found the guinea-pig inoculations so time consuming as to be impracticable as a routine procedure. In the series of sixty patients submitted to both tests, the diagnosis was in most instances made on a careful study of clinical symptoms, cystoscopy and smear examinations.

In examining the kidneys of the guinea-pig after it has been killed, Medlar and Sasano point out the necessity of making serial sections of both kidneys to avoid overlooking tuberculous lesions that may have developed there. They conclude that negative findings do not rule out tuberculosis. Incidentally, they found no evidence to support the theory of excretion of tubercle bacilli from a normal kidney.

CYSTOSCOPIC EXAMINATION

Bladder Appearance. The changes observed in the bladder in cases of renal tuberculosis are so characteristic that one who is familiar with them can almost make the diagnosis at a glance. Two types of lesions exist: granulations and ulcerations of the bladder mucosa.

The granulations, which are about the size of the head of a pin, may be single or multiple. In the latter case they may form a cluster or ring around the ureteral orifice, the trigone or the fundus of the bladder. Each is a small pustule of a yellowish color surrounded with a red halo.

The ulcerations, too, fall into two classes, some being slightly elevated with red margins, the others deeper, somewhat more extensive, with their margins notched and eroded and exhibiting a variety of colors. At times they are difficult to distinguish from ordinary ulcerations which may also be present at the same time. If one or both of the ureteral orifices are seen to be rigid and gaping, the picture should immediately suggest tuberculosis. With time the bladder contracts and its capacity diminishes. The deformities of the ureteral orifices cause a deviation of the trigone which is pulled out of shape by the rigid and contracting ureters. Trigonitis is pronounced and edema develops at the bladder neck.

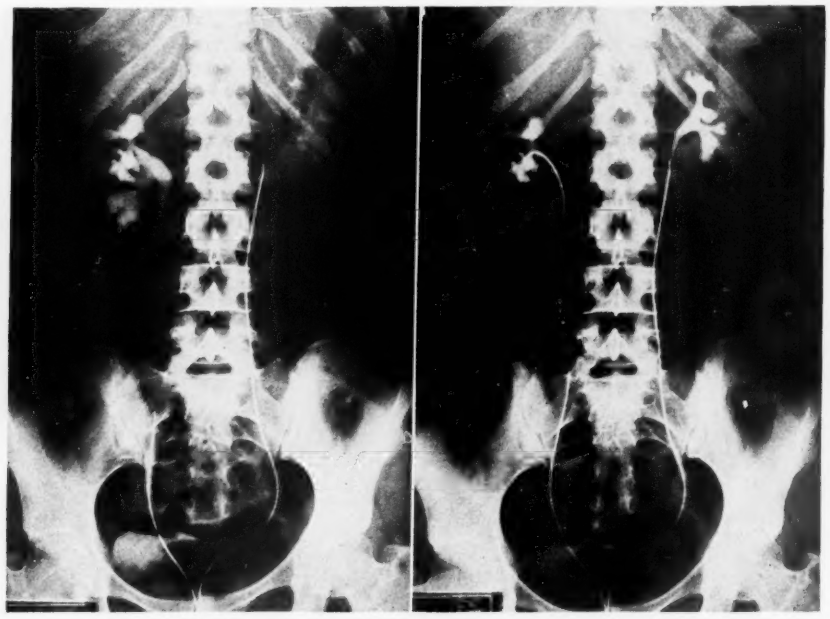
DIFFERENTIAL DETERMINATION OF KIDNEY FUNCTION

In determining what course to pursue in treatment of a tuberculous kidney, it is of the first importance to know the condition of both kidneys as regards function and the amount of infection present in each. By ureteral catheterization we are able to localize the site of the tuberculous process, to know whether tubercle bacilli are present in one, or both, kidneys, to determine the extent to which each kidney is excreting pus and blood cells, and the ability of each kidney to excrete indigocarmine or phenolsulfonphthalein.

In bilateral renal tuberculosis the answers to these tests may save the life or speak the doom of the patient. The question arises as to the criterion on which to base the diagnosis of bilateral renal involvement. We have seen that some surgeons will not remove a kidney if its mate is found to contain a single tubercle bacillus, even though there is no sign of blood or pus, and all functional tests are satisfactory. Hyman and Mann, Braasch and others, point out the doubtful wisdom of considering a kidney tuberculous under such circumstances, when such a decision may spell death for the patient who is denied the benefit of a needed nephrectomy. They remind us that the mere presence of pus in the second kidney does not prove tuberculosis for it may be due to a non-specific infection. The combined presence of the tubercle bacillus, of pus and of blood in the urine of the kidney under consideration would seem to be a minimum requirement for a diagnosis of tuberculosis in that kidney. The bacilli, without pus or blood, in a clear urine, may be simply a contamination picked up by a catheter passing through the bladder or due to reflux. The diagnosis of each kidney

should be made on *all* the data, not a technical failure or one rather arbitrary criterion.

renal parenchyma becomes necrotic in areas producing an irregular dilatation of the pelvis or calices. (Fig. 2.) Generally



A B

FIG. 1. A, right retrograde pyelogram. Cortical necrosis of the upper and middle calices with irregular dilatation of the pelvis and lower calyx. B, left retrograde pyelogram. Normal pelvis and calices. (Urine negative from this kidney.)

UROGRAPHY

A small defect in the pyelogram of a patient with an essentially negative bladder may direct the examiners attention to the possibility of a tuberculous infection.

Braasch has most ably described the most important changes caused by tuberculosis in the renal pelvis and ureter as follows:

1. *Areas of Cortical Necrosis in the Kidney.* This defect will usually be seen in the outline of one or more of the calices and the appearance is that of irregular dilatation with uneven "moth-eaten" borders. (Fig. 1A and B.)

The apices of the calices are more likely to show this deformity with occasionally detached areas in the adjacent renal parenchyma. Unless the pathologic process involves the renal papilla no deformity characteristic of the disease will be obtained in the pyelogram.

2. *Irregular Dilatation of the Renal Pelvis.* As the inflammatory process continues the the renal pelvis is not tremendously enlarged unless stricture has developed in the ureter and produced some degree of hydronephrosis.

Braasch further points out that when the process is confined largely to the cortex, the pelvis may become contracted in a manner similar to certain forms of atrophic pyelonephritis.

3. *Stricture of the Ureter.* The infection produces areas of ulceration in the ureter which in time become cicatricial and result in a definite stricture. These areas of constriction are usually multiple and appear urographically as alternating areas of constriction and dilatation.

Crenshaw found 7.1 per cent of the cases of renal tuberculosis under his observation to have calcification and found this condition more common in men. The calcification is cortical, irregular and may be miliary. (Fig. 3.)

In considering a pyeloureterogram one may be confused by certain deformities

produced by pyelonephritis and artifacts. In pyelonephritis the dilatation is more commonly uniform in its involvement.

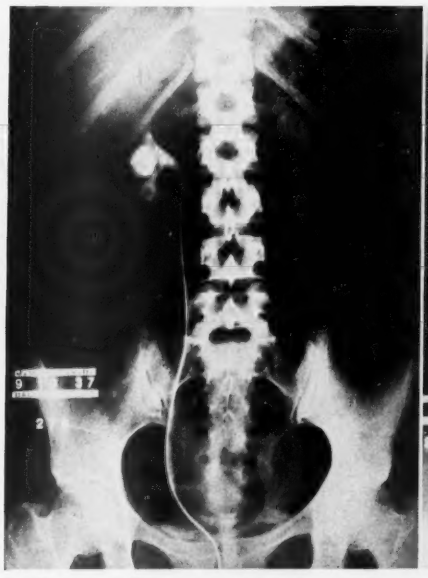


Fig. 2. Right retrograde pyelogram. Irregular dilatation of the pelvis, upper and middle calices.

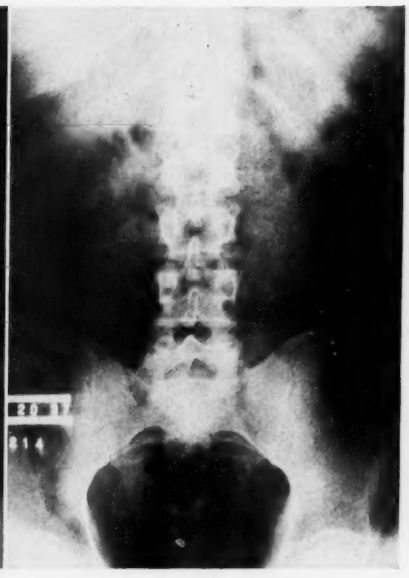


Fig. 3. K.U.B. Triangular area of calcification in the mid-cortical portion of the left kidney. Irregular areas of cortical calcification in left kidney. (Acid fast organisms recovered from each kidney.)

Artifacts may be produced by overdistention and incomplete filling of the renal pelvis with the pyelographic medium. The extravasation distal to the tips of the calices resulting from overdistention, when associated with pyelonephritis, may appear similar to the deformity produced by cortical necrosis as a result of tuberculosis. Incomplete filling of the pyelogram may likewise result in confusion in the interpretation.

Excretory urograms, as a rule, do not demonstrate the early defects characteristic of renal tuberculosis. However, the excretory urogram is valuable in following the course of the disease once the diagnosis has been established and it gives additional information as to the function of the two kidneys. Evidence of gross disproportion in the involvement of the two kidneys is clearly demonstrated by this method.

SUMMARY

The incidence of renal tuberculosis is today less than in the past two decades due to the reduction in the incidence of other forms of tuberculosis. It has been fully established that renal tuberculosis is secondary to tuberculous infection elsewhere in the body and that the organism is carried to the kidneys by the blood stream in most instances.

The question of bilateral involvement of both kidneys, at the outset, is not fully decided; however, the work of Medlar and Sasana demonstrated 88 per cent of their guinea-pigs to have bilateral renal lesions. Clinically, Braasch and Sutton were only able to demonstrate 291 cases (13 per cent) of bilateral involvement in a total of 2,200 cases of renal tuberculosis studied.

The symptoms vary from nil to those of a very painful cystitis with later pain and tenderness in the kidney region. Occasionally, hematuria may be the first symptom. A deceptive feature in some cases is the occurrence of long remissions free of painful symptoms. No symptom or train of symptoms is pathognomonic of renal tuberculosis.

The urinary findings give the most important evidence of the presence of renal

tuberculosis. Pus cells are regularly found and red blood cells and albumin are usually present throughout the entire course of the disease. The acid fast bacillus is found on staining the centrifuged urinary sediment in a large percentage of the cases and guinea-pig inoculations are to be employed when the smears give no decisive results.

The cystoscopic appearance of the bladder is quite characteristic. Two types of lesions exist: granulations and ulceration. The function of each kidney is to be determined as well as the microscopic contents in the urine of each kidney. The presence of a few organisms in the urine from one kidney in the absence of pus cells, red blood cells and albumin may represent a contamination in passing the ureteral catheter through an infected bladder.

The pyelogram is of great value. A small defect may first direct the examiner's attention to the possibility of a tuberculous infection. Cortical necrosis is demonstrated when the lesion involves the renal papilla. The excretory urogram usually does not demonstrate early defects as well as the retrograde pyelogram. However, the excretory urogram is valuable in following the course of the disease and in demonstrating disproportion in the involvement of the kidneys.

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TUMORS OF THE KIDNEY*

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THAT individual presenting himself for urologic care which subsequently unfolds the probability of renal tumor rarely does so because of an awareness of his disease but because of a symptom (or group of symptoms) which to him is sufficiently divergent from his customary health to arouse his desire for medical sanctuary. The degree and duration of divergence necessary to establish this desire, coupled with the acumen of the medical advice received prior to urologic examination, frequently but not always determine at what phase in its biologic cycle the tumor is to be attacked. While it is possible that the public has been unwittingly educated into expecting *too* great a panacea from early diagnosis, one may scarcely deny that the eventual outcome is influenced in a large degree by the distance from its inception at which the tumor is treated. It thus becomes the obligation of the profession at large to suspect the possibility and direct the proper investigation before all classical symptoms and findings have developed, as well as the duty of the urologist to perfect the technic of early diagnosis and the improvement of treatment leading to a future of more hope than the gloom of the past.

While in practice the problem is that of synthesizing the component parts of history, physical examination, laboratory and roentgen findings into diagnosis and treatment, it is, *per se*, in a written discussion of the subject, necessary to start with acceptance of the fact, a tumor, and thence, in the opposite direction, by analysis to develop those various components. The absence of any pathognomonic picture, at least until late in the life of the disease, does not facilitate the problem. It is further unfortunate that our knowledge of pathological conditions and treatment is far from the complete agreement of the recognized authorities whose honest differences of opinion are greater on these two aspects than in the problems pertaining to symptoms, findings or diagnosis. It is our undertaking at present to present a constructive clinical review of renal tumors, but it must be admitted with any candor at all that even the most casual survey of the literature is bound to reveal certain controversial points relating to pathological conditions (including classification) and treatment. Happy is he who could maintain a view of renal tumors thoroughly harmonious in all aspects with all recognized authorities. If one wishes to enter the controversy, he should repair to the special articles in the literature rather than to standard texts.

It is not in our domain to consider here cysts of the kidney, whether solitary or multiple, nor cyst-like enlargement due to hydronephrosis or pyonephrosis. They are not in the category of new growths, nor is congenital polycystic disease of the kidneys which, though it may not become patent till adult life, falls more customarily under the heading of congenital anomalies.

EMBRYOMA

There are six comprehensive contemporary American textbooks of urology. Half of these present a classification on the first page of the chapter on renal tumors, the others implying one by discussion of the subject under topical headings or including it later in the chapter. For the most part those tumors of the kidney occurring in the first decade of life receive

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separate consideration from those of mature years. These tumors are not *frequently* encountered outside of a children's hospital or a large medical center. It is not our intent to include their comprehensive discussion. They do, however, constitute the most frequently encountered malignant abdominal tumor of childhood. The embryoma, or tumor of Wilms, reveals itself usually by the development of a large abdominal mass in the renal area. Hematuria is not an early symptom. Pain and cachexia may or may not be present. Embryoma should be suspected until excluded in every child with a solid mass in the renal fossa. Hypertension has been noted by Koons[5] in a girl of seven years, and relieved by nephrectomy. The patient died (since the publication of his paper) of an hepatic metastasis.* Metastasis occurs after the tumor has obtained considerable size. Diagnosis rests upon detection of the mass and pyelography. Abdominal palpation should be restricted and not indiscriminately repeated because of the danger of metastasis therefrom.[7] Urinalysis and other routine laboratory analyses will probably be of little aid.

Excretory urography may, under very rare circumstances, obviate further urologic study but it is probably true that no nephrectomy should ever be performed on child or adult *solely* on the basis of findings thereby derived. Retrograde studies are necessary to confirm the presence, condition and function of the other kidney before sacrifice of the condemned one, as well as for diagnostic purposes. On the other hand, anteroposterior retrograde pyelographic study alone is inadequate if not positively diagnostic and should be coupled with a lateral pyelogram as well.

Treatment, insofar as there is agreement, is first preoperative irradiation, and second, nephrectomy (if metastases are not present), followed by postoperative irradiation. Diagnosis is confirmed by gross and histologic examination of the tumor, which will reveal cells originating from types of more than one germinal layer. There is a general trend in favor of the transperitoneal route if the tumor is of large size. This affords better exposure as well as an opportunity to ligate the pedicle before the mass is traumatized incidental to its dissection. The capsule and perirenal fat should be removed intact with the mass so as to include undemonstrable extension and lymph-nodes. The prognosis is almost universally recognized to be bad, death ensuing from six months to two years after operation as a rule.

It is well here, however, to call especial attention to the report of Ladd[7] who gives the characteristics of an embryoma as follows: variegated histology, rapid growth after a period of slow activity, hemorrhage and necrosis perhaps leading to capsular rupture, possible invasion of pelvis or vein, metastases after attaining large size, and frequency of local recurrence. Ladd does not advise routine retrograde catheterization because of the delay involved, nor does he favor preoperative irradiation because of its failure to kill the entire tumor. He raises the question as to whether the latter prevents early dissemination. He favors immediate transperitoneal nephrectomy. His operative mortality is 7 per cent, and of forty-five patients he reports fourteen alive, eleven of whom are probably cured one and one-half to nineteen and one-half years postoperatively. We have once observed ill effects from irradiation on the good kidney. Examination revealed no evidence of metastases or renal failure. After partial completion of irradiation the left kidney decreased 50 per cent in size. Completion of irradiation resulted in no further decrease but was accompanied by beginning evidence of nitrogen retention and renal failure. In three months autopsy showed a well walled-off left kidney whose histology was of embryonal type. The right kidney was contracted, sclerotic and showed abnormal fibrosis. There were no metastases. Our suspicions could hardly fail to be roused as to a possible relationship between *x*-ray therapy and au-

* Personal communication.

topsy findings. Priestley[11] uses preoperative irradiation, nephrectomy and postoperative irradiation over thorax, renal area and abdomen. Of thirty-seven patients with embryomas he reports 33 per cent alive more than three years and 9 per cent more than ten years after operation.

PARENCHYMAL TUMORS OF ADULT LIFE

There is no sufficiently characteristic symptom complex of renal tumors of adult life to draw a typical picture. It is rather a partial disclosure of the whole which should constantly be in the mind of the physician. Rarely is such a tumor detected as a result of an accidental, suggestive finding in the course of a routine examination. More often it is because of complaint of the patient due to a symptom originating from a change in the size or position of the kidney, from an abnormality created in the drainage system of the kidney or from an unusual appearance of the urine. In a few instances symptoms have been reported developing from metastases while the home growth was too small to focus any attention. If we anticipate the classical picture of mass, pain and hematuria, we may have to wait a long time in the life of the disease, or be misled (as we recently were when we exposed a polycystic kidney) by some other finding.

UNUSUAL TUMORS

Before entering a detailed discussion of the problem we must limit and classify it. Clinically, benign tumors are so rare that we must forego their review. We have seen a benign adenoma incidentally reported by the pathologist in examining a kidney removed because of hydronephrosis. In another instance a calculous pyonephrosis incidentally showed so-called benign papillomatosis of the renal pelvis. The patient is alive and well nine years later. Non-epithelial tumors, including those of vascular origin, are also rare. Metastatic tumors from elsewhere which situate themselves in the kidney are also rare. Abeshouse and Goldstein[1] found that carcinoma in this connection is five times as frequent as sarcoma; but *relatively* speaking, because of the higher incidence of the former as a primary lesion, sarcoma is twice as common as carcinoma. The kidney is never the solitary site of metastasis. The other kidney is most frequently the original focus. Once we observed such an instance. A highly malignant "adenocarcinoma of renal epithelium" of the left kidney was thought by the pathologist to have metastasized to the right kidney as well as elsewhere (autopsy). In another, a carcinoma of the bladder was found to have involved the right kidney. In a third, a similar primary tumor metastasized to the right suprarenal gland, and at the time of autopsy appeared to be invading the adjacent kidney by extension. Two different primary types of tumor in the same kidney are probably the rarest combination of all.

TUMORS OF FREQUENT INCIDENCE

We are all indebted to Hertzler for the statement that solid tumors of the kidney represent ½ per cent of all tumors. They are usually malignant though the degree may be one of variation. We must recognize two gross types, namely, those arising in the parenchyma and those in the renal pelvis, with subdivisions of each. It is in the subdivisions that differences of opinion arise.

Classification: The most frequent tumor of the adult kidney is the hypernephroma. Grawitz[3] believed they developed from adrenal cell rests. Sudeck and Stoerk[3] thought they were adenomas arising in the kidney tissue. Ewing[3] and most contemporary authorities now believe that the majority of malignant epithelial parenchymal tumors are true carcinomas and not derived from adrenal tissue rests. The term hypernephroma, however, is one of tenacious clinical tradition and cannot be shaken by our present day knowledge, regardless of what school we follow. We find that there is about an equally divided tendency of current authors to refer to epithelial malignant parenchymal tumors

as carcinoma or adenocarcinoma on the one hand, and on the other, to recognize the hypernephroma (of indeterminate origin) as an encapsulated, slowly progressing tumor of a lesser degree of malignancy, and the adenocarcinoma as a wildly growing, infiltrating, highly malignant tumor. The first mentioned school subdivides carcinoma into three groups: the papillary adenocarcinoma, the alveolar adenocarcinoma and the malignant papillary cystadenoma. Hypernephroma is more or less encapsulated and apt to lie at one pole of the kidney. On section it may have yellowish streaks and show evidence of hemorrhage, necrosis or cyst formation. The adenocarcinoma is gray or whitish, not encapsulated and fails to reveal necrosis or hemorrhage.

Kozoll and Kirshbaum[6] use the terms, benign or malignant hypernephroma, both of which are yellow and vascular, with a central fibrous core and a cellular cortical area with histologic structures similar to the fasicular zone of the adrenal cortex. The benign ones are small, well encapsulated, produce no deformity, do not metastasize, and present no diagnostic clinical features, always being discovered at autopsy. These they call the benign hypernephroma or hypernephroid adenoma. The malignant or hypernephroid carcinoma are grossly malignant, larger and run a clinically malignant course. In 12,885 consecutive autopsies they found thirty-three of the former and forty-four of the latter. In some which were histologically benign, distant metastases were found. Hence they studied only autopsy material to verify the presence or absence of metastases as an aid to establish criteria of benignity or malignancy. In either instance males predominated 4 to 1 and whites to blacks 4 to 1. Most of their patients representing malignant cases were unoperated upon because they presented themselves too late. Only six of the forty-four died without metastases, the lungs being the most frequent and the bones the least frequent sites. Only five lived over one year. Six of the malignant ones were of the transitional type, grossly benign but invading the capsule and parenchyma. It is unfortunate that it is clinically impossible to adopt the biologic methods of study of Lucké[8] to which the reader is urged to refer. The fact remains, however, that while the pathologist may discover these relatively benign epithelial parenchymal tumors, in practice the life cycle of the growth has usually advanced beyond this point. We have observed it only twice and that at autopsy. Both were prostatics whose autopsy findings revealed hitherto unsuspected hypernephroma which were in no way related to the cause of death.

Clinical Aspects. We have observed in our group (Hamer, Mertz and author) thirty-five patients with malignant tumors of the kidney since 1928. None were seen solely by the author. All were private patients with three exceptions included for special interest. Five of these will be discussed later under the subject of renal pelvic tumors. All of these diagnoses were verified by operation, exploration with biopsy or autopsy. Of the thirty parenchymal tumors two proposed nephrectomies terminated in unsuccessful exploration. One of these was verified later at autopsy. The other was verified by biopsy due to inadvertent removal of a piece of tumor from the capsular region. The former lived eight years after unsuccessful exploration. He received postoperative irradiation. Autopsy revealed adenocarcinoma. The latter was lost to study. Hers was a hypernephroma. Twenty-four of the thirty parenchymal tumors were hypernephroma, four were adenocarcinoma, one was a sarcoma and one a metastatic tumor from the bladder which was unsuspected.

Symptoms. The patient will probably be a white man between forty and sixty-five years of age. He may complain of any or all of the common symptoms. Pain is at times of a dull character, located in the costovertebral angle or upper abdomen, perhaps due to capsular tension. Gillies[4] found that fifteen of seventeen such in-

stances were accompanied by a palpable mass. Severe pain or colic is less frequent and is due to an interference with the drainage of urine from the pelvis or ureter. This may be the result of a mechanical pressure effect or, more likely, to the passage of a clot. Pain due to metastases will be predicated upon the location of the latter. Pain is not a diagnostic symptom in itself.

Hematuria frequently is present but not always. Its absence does not exclude the disease. It may be macroscopic or microscopic. In the latter event it will of course be the physician's discovery. The number of urinalyses terminating without microscopic examination continues to be appalling and is due to apathy rather than ignorance. Worse than this, even in the year 1941, emphasis on the importance of gross hematuria still continues to be a missionary obligation. Of all aberrations from normal bodily functions gross hematuria would seem to be as striking and dramatic as any. Whatever its cause it is almost always self limited, in its initial phases at least. For the patient it is perhaps natural to grasp at the hope of vis naturae medicatrix; for the physician it never should be. Because of this early self limitation it, therefore, follows that hematuria will stop because of or in spite of whatever medication is tried. It always demands anatomic investigation as to its origin. *Then* let one use what nostrum he will as long as it does not delay proper treatment. In the absence of acute gonorrhea, nephritis or some other obvious and certain medical cause, one subsided attack of gross hematuria always remains as important an indication for special investigation as continuous bleeding. Otherwise we shall continue to see such horrible examples as gross hematuria of sixteen years' duration due to neoplasm of the urinary tract on the assumption of Bright's disease. With hypernephroma, hematuria occurs as a rule from hyperemia or when the tumor communicates with the pelvis or a calyx. It is not as profound as in renal pelvic tumors. Pyuria occurs in the face of secondary infection.

The tumor is palpable earlier when the growth is in the lower pole. Anterior palpation is insufficient. A hand should be placed in the loin and another on the anterior wall with gradual but deep pressure during several respiratory cycles. Unless the growth has become fixed it will move with respiration and be ballottable. It may be smooth or bosselated. If bilateral the thought would be polycystic disease. In embryoma, the patient or his mother will mention the tumor. In hypernephroma the responsibility is more apt to be up to the physician to find it, though not always.

Anemia is more often due to toxemia than actual loss of blood. Fever is an occasional finding. Hypertension, in the light of recent knowledge of its relation to the kidney, is interesting but the causal relationship unsettled. We have not observed its permanent reduction following nephrectomy. Koons, as previously mentioned, has, and this to date remains the only example in Page's clinic. Spontaneous perirenal hemorrhage is rare and still more rarely diagnosed unless encountered more than once by the same physician.[14]

Preoperative diagnosis ultimately rests on urography. Nichols[10] believes that, although more difficult to interpret, excretory urograms are of great value. If used routinely on all admissions, it would doubtless detect some renal tumors which would otherwise escape. Used exclusively on suspected cases our fear would be for the converse. Waters[12] does not advise it. Certain it is that unless the so-called negative excretory urogram is technically perfect (i.e., well filled and well visualized), it should be verified by a retrograde pyelogram before the uncertain case is freed of its suspicion. It is also frequently essential to know more about the other kidney (presence, function and freedom from infection or other defect) than excretory urography may tell. The original roentgenogram should be as free from gas as possible to delineate clearly the renal out-

line. In hypernephroma this might be enlarged or irregular. Braasch[13] has called our attention to occasional findings of calcification which he considers a good omen.

Cystoscopic examination may reveal the side from which the bleeding occurred. Blood, however, in ureteral specimens of urine is of no special diagnostic import (trauma). Special examination also should give us comforting data previously mentioned about the other kidney. As to the pyelogram itself the frequently quoted term "filling defect" requires some explanation. This condition is present in classical examples in tumors of the renal pelvis and clot or nonopaque stone in the pelvis. In the case of hypernephroma, if the tumor has invaded the pelvis or if by pressure it has occluded a calyx or infundibulum, the "filling defect" will be there. In other events we may have rather a bizzare type of pelvis characterized by distortion or displacement. The other pelvis should be normal. Instances of doubt may be clarified by the addition of a lateral pyelogram as Mertz[8a] has clearly demonstrated. Gillies[4] has observed the retrograde pyelogram to show as follows: Cortical elongation and compression in 60 per cent; pelvis and calyx pushed to one side; pelvic invasion uncommon; complete obliteration of pelvic and cortical landmarks in 25 per cent; elongation of calyces with no compression but with dilation in 7½ per cent; normal pyelograms in 0 per cent. In differential diagnosis our own experience has revealed our shortcomings must make us most alert to rule out polycystic kidney, renal tuberculosis, retained clot in the pelvis and extrarenal retroperitoneal tumor with possible renal invasion.

Treatment again leads us into some controversial issues. Barringer[2] has stated that from his own cases irradiation of small doses over a long period gives better results than irradiation followed by operation. Munger[9] favors ultra short wave preoperative irradiation, transperitoneal nephrectomy and limited postoperative irradiation. Waters[12] finds that over 90 per cent of cortical tumors are radiosensitive, exclusive of papillary cystadenoma (and of course papillary carcinoma of the pelvis). Nephrectomy should follow in two months. Munger does not delay over a fortnight. Braasch believes that irradiation may not reduce the size of the tumor and may increase its fixation and, therefore, is not always of help. Priestley[11] advises preoperative irradiation of the very large tumors to facilitate nephrectomy. He uses the lumbar approach except in very large tumors. Our own experience with irradiation has been limited. We have used it for recurrences which it may have delayed because one such patient was known to be alive eight years and the other three years after operation. An inoperable patient (adenocarcinoma) whom we explored received postoperative irradiation, and again seven years later, shortly after which he died. We know of another instance, however, in which unsuccessful exploration was not followed by irradiation but life was the same duration.

It is almost universally conceded that operation is essential. The present trend favors the transperitoneal approach for reasons mentioned in discussion of embryoma. Pre- or postoperative transfusion may be needed. Contraindications to operation are fixation, metastases, age, cachexia and infection.[13] The other kidney must of course be able to sustain life. Metastases from hypernephromas, though late in the cycle of the disease, are notoriously frequent and generally contraindicate operation. Metastases are chiefly to the lungs, liver and bones. Hypernephroma causes osteolytic osseous metastases and is one of the four most frequent tumors causing bony metastasis (prostate, thyroid and breast). Barney has reported a patient apparently cured by nephrectomy and lobectomy for pulmonary metastasis.

Prognosis depends upon the promptness of diagnosis after onset of the disease, on the size of the tumor, on metastasis, on fixation of the tumor, on histogenesis and

on other factors associated with the general condition of the patient. Some believe that the gradation of the degree of malignancy of the tumor has paramount bearing on prognosis (Priestley, Broders, Braasch, et al.) while others do not. Our own experience has been that, with a few gross exceptions, there is relationship. Six and two-tenths per cent of sixty-four patients reported by Waters lived five years without demonstrable recurrence or metastasis. Braasch[13] reports 42 per cent alive over five years after nephrectomy. With explored but unremoved kidneys, 17 per cent were alive for a similar period. Priestley[11] finds the greatest mortality in the first three years after operation. After this, those who succumb do so according to mortality figures of the general population of a similar age. Seventy-three per cent of the patients in his report with grade 1 adenocarcinoma of the renal parenchyma were alive three or more years after operation, 22 per cent with grade 4; 47 per cent with grade 1 were alive ten years or more and 3.2 per cent with grade 4. Forty-six per cent of his patients reported with tumors weighing less than 500 Gm. lived five years or more and 24 per cent with tumors weighing over 1,000 Gm. For more extended study of his very informing report one should consult his article listed in the bibliography.

Of our own twenty-four patients with hypernephromas two may be excluded because of their incidental autopsy finding with death from another cause. Of the remaining twenty-two one was inoperable but explored, three were operated upon elsewhere and showed evidence of recurrence. Of the remaining eighteen we know of five deaths (two months, two, three, five and ten years postoperatively). One of those who died had a spontaneous pathologic fracture, the only such instance we have observed. Only two were due directly to the tumor (local recurrences and metastasis). Of the eighteen, there were four known recurrences (three still alive). Six others are known to be alive and well; one 1 year after operation (and she had a tumor thrombosis in the renal vein), one four years, three six years and one ten years. Five were poorly followed but two of these were known to be alive and well one year after operation. Benign hypertrophy of the prostate was the most frequently encountered, associated but unrelated disease.

Not included above is one patient with a sarcoma alive one year after operation. Four of our patients have had adenocarcinoma. One metastasized to the other kidney as revealed by autopsy (patient not operated upon). Two were operated upon, one of whom had an alveolar adenocarcinoma of the parenchyma associated with calculus. The other had ascites nine years after operation, probably due to recurrence. He was lost sight of before death. The fourth had an unsuccessful exploration and died eight years later.

Our own results with parenchymal epithelial tumors of the kidney would thus lead us to conclude that the adenocarcinoma is more malignant than the so-called hypernephroma and apt to be more inoperable. Preoperative differentiation was not made. Cortical tumor has not been encountered by us in enormous size except once in a private patient and, therefore, transperitoneal removal has not been employed nor has preoperative irradiation been routinely necessary in order to make the tumor removable. Operative death in the hospital was not encountered in our small series of hypernephromas which would lead us to conclude, probably erroneously, that mortality is no greater than from nephrectomy for other causes. Our deaths will begin within two months after operation. Recurrence and metastasis are not uncommon but the incidence of bony metastasis was low.

RENAL PELVIC TUMORS

Epithelial tumors of the renal pelvis are thought to be rather infrequent, comprising from 3 to 20 per cent of all renal neoplasms. Gillies[4] found them in 10 per cent of his series. We have noted them in five of

thirty-five patients with some sort of renal neoplasm. This is considerably higher than the average which is generally stated from 5 to 7 per cent. We have found three-fifths to be of the papillary type which conforms to most reports. Lowsley states that men are afflicted with papillary tumors more often than women, which has been our experience. Gillies found no sex difference. Four of his five patients were seventy or over. Three-fifths of our patients also were over seventy. We have observed only one so-called benign pelvic tumor (papillomatosis) but have not included it in this series because the patient was operated upon for stone and the tumor was an incidental finding.

The nonpapillary flat types (squamous cell) are reputed to develop more often than the papillary types in the presence of stone, leukoplakia or chronic inflammation. Nonepithelial types are rare. Renal pelvic tumors associated with cortical tumors are still more so. Balch, of Indianapolis, has encountered one instance of papillary carcinoma of the pelvis in a kidney harboring a hypernephroma. Pelvic tumors may invade the parenchyma. They cause hydronephrosis more often than cortical tumors. They spread via the lymphatics. Metastases also spread to the lungs, liver, adrenal and bone (Lowsley). The papillary type is prone to occur coincidentally in the ureter and around the ureteral orifice in the bladder, not, according to Keyes, because of implants but because of the same factor which prompts the origin of the growth in the pelvis. The prognosis is absolutely bad in the nonpapillary type and better in the latter because it metastasizes later.

The signs of the disease are similar to those of cortical tumors but vary in certain respects. Hematuria is more intermittent and severe. Clots are frequent. Pain is due to obstruction of the pelvis or the passage of clots. Tumor mass is less frequent. Tissue may be found in the urine and loss of weight and anemia are not uncommon.

Preoperative diagnosis is difficult in differentiating from cortical tumors and rests on history, physical and special urologic examinations. The last mentioned consists of (1) urinalysis which may reveal tissue cells; (2) cystoscopic examination which may show bladder involvement and which is apt to provoke traumatic bleeding when the pelvis is catheterized and (3) pyelography which discloses either hydronephrosis, distortion, true filling defect or a combination of all. Clots and nonopaque calculus must be excluded in differentiation of the pyelogram.

Treatment consists of nephroureterectomy unless contraindications (see hypernephroma) are present. The tumor is radioresistant and irradiation is, therefore, not as much avail. The kidney and upper ureter may be removed by the lumbar route and the lower ureter together with a sufficient cuff of bladder wall through an anterior extraperitoneal incision.

Prognosis of pelvic tumors is not good due to the highly malignant type of the tumor, to the possible presence of perirenal extension and to metastases. The fact that these tumors occur at an older age of life than the cortical tumors does not enhance the patient's operative risk. Braasch states the average length of life as less than two years. The papillary type offers better prognosis than the flat variety, no five-year cures of the latter being on record (Lowsley). Priestley reports 45 per cent of patients with pelvic tumors alive three or more years after operation, 35 per cent for five or more years and 10 per cent for ten or more years.

Our own contact with renal pelvic tumors follows: Correct diagnosis was established before pathologic diagnosis in two of five patients. A woman of seventy-seven years, complaining of pain in the left renal fossa, had a pyelographic diagnosis of hypernephroma. Subsequently, squamous cell carcinoma was found but our records do not make clear whether this was based on autopsy or operation. She died within several months of the first diagnosis, hav-

ing been treated by irradiation before correct diagnosis was made.

A man, sixty-two, had a preoperative diagnosis of calculous pyonephrosis. His complaints were hematuria and gastrointestinal upsets. Findings at operation showed duplication of the pelvis as far as the ureteropelvic juncture, with a stone at that point, and pyonephrosis. In the course of nephrectomy a tumor of the lower pelvis and lower pole of the kidney was found. The tumor had extended into the cortex and the perirenal fat, as much of which as possible was removed. Pathologic diagnosis pertaining to the neoplasm revealed a transitional cell carcinoma of the pelvis with invasion and penetration of the kidney. Four months later the patient had "resistance in the left abdomen" but no mass. He died five months after operation of recurrence or incomplete removal. Irradiation was given after the patient returned home, ending two months before death. He improved clinically during the period.

A third patient was a man, fifty-six, with hematuria whose preoperative diagnosis was hypernephroma. He had a pathologic diagnosis of grade 2 papillary carcinoma and a solitary cyst. However, we had treated him ten months thinking the bleeding to be from the prostatic hypertrophy before renal tumor was found. Urologic examination five months after operation showed no bladder extension. He received preoperative irradiation, largely because hypernephroma was suspected. He is now well six months after operation without evidence of recurrence or metastasis.

A colored man of seventy-four complained of hematuria. Small benign hypertrophy of the prostate was found which bled on trauma and was resected. Hematuria continued so an upper tract investigation was made in one month. Left duplication of pelvis and ureter was found; hematuria was on the right. Hydronephrosis with filling defect prompted a preoperative diagnosis of papillary carcinoma of the pelvis. Lumbar nephrectomy was done, the pelvis torn accidentally and the incision contaminated with cauliflower-like tumor. There was no evidence of perirenal involvement. The incision was filled with tincture of merthiolate and sponged thoroughly before and after. Intensive postoperative irradiation was given. Pathologic diagnosis was papillary squamous cell epithelioma of the pelvis, hydronephrosis, ureteritis cystica, and fibroma of the medulla. No recurrence has developed to date (twenty months after operation). The upper ureter was removed with the kidney and showed no neoplasm. Complete ureterectomy was not done because it was believed at the time that the patient would not stand it.

The last patient was a woman, aged seventy, who had been treated seven years for right pyelitis. Hematuria then developed and there was a change in the pyelographic findings with hydronephrosis and filling defect. Lumbar nephrectomy was performed with a diagnosis of tumor of the pelvis. The pathologic report showed malignant carcinoma of the pelvis with papillary formation, hydronephrosis and bifid pelvis. Cardiorenal death followed in ten years. Autopsy showed pyelonephritis of the remaining kidney and multiple vesical diverticula with stones, no recurrence and no metastasis.

In our four operative cases mistaken diagnosis in two did not suggest ureterectomy. One of these was in too poor a condition to have tolerated it if contemplated, and would have died at any rate because of perirenal extension, even if ureterectomy had been done. The other has no recurrence but is only six months post-operative. In the two patients with correct preoperative diagnosis the age and feeble condition restrained us from ureterectomy. One died ten years after nephrectomy without recurrence and the other is alive twenty months after operation and in good health. We do not mean to imply that ureteronephrectomy is not warranted but rather that we happen to have been fortunate. None of the patients showed ureteral extension histologically. Pelvic

infection was prominent in all four operative cases. There is an obvious lesson from two of the three male operative cases, viz., hematuria assumed to be due to benign hypertrophy of the prostate, which does not stop promptly with proper treatment, should at once indicate investigation of the upper urinary tract. Hematuria without a palpable kidney was constant in all four patients. With elderly patients of long standing upper tract infection, periodic pyelography should be performed to detect the onset of any new development such as carcinoma of the pelvis.

Extrarenal retroperitoneal tumors[14] are a chapter in themselves and will not be discussed here. They may, however, be confusing from point of view of differential diagnosis and should be distinguished from cortical or pelvic tumors.

SUMMARY

The difficulties of the various problems of renal neoplasms are discussed. Embryonal tumors of childhood are touched upon. Rare parenchymal tumors are mentioned by name only. Most frequently encountered parenchymal tumors ("hypernephroma" and adenocarcinoma) are discussed with minimal pathologic consideration, and at some length clinically from point of view of symptoms, diagnosis, treatment (operation and irradiation), prognosis, and our own experiences (including blunders) with thirty patients. Tumors of the renal pelvis are discussed in a similar manner with relatively more emphasis on our own findings in five patients.

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CANCER OF THE BLADDER

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THIS paper is primarily intended to include some of the pertinent points regarding the diagnosis of cancer of the urinary bladder. The satisfactory treatment of vesical malignancies, not unlike the majority of cancer involving other parts of the body, depends for the most part upon early diagnosis and prompt institution of adequate therapy.

The writers believe that it is of vital importance to adhere to a rigid schema and preoperative program for studying each suspected case. Insufficient information is acquired from cystoscopic examination alone. Emphasis must be placed on the following points: (1) history of the case, (2) physical examination, (3) urinalysis, (4) classification, (5) cystoscopy and biopsy, (6) cystography and urography, and (7) differential diagnosis.

History. A careful history is of considerable importance because the duration of symptoms and the severity of symptoms may give one an index as to the degree of malignancy. This is shown by the fact that painless hematuria of an intermittent type is often the only complaint and it may have been noticed and ignored by the patient for a great many years. We know of one case in which the patient had gross hematuria eighteen years prior to seeking medical counsel. At operation a large papillary carcinoma with infiltration of the bladder wall was found. Had this patient sought advice with the advent of his initial hematuria it is probable that a thorough transurethral destruction of the tumor may have resulted in a permanent cure.

From the histories of patients with bladder neoplasm it will be learned that in a very high percentage of the cases the initial symptom was gross hematuria. In reviewing our material for study which consists of 211 cases of malignancy of the bladder examined and treated at the Mercy and Presbyterian Hospitals in Pittsburgh, we learned that, in 146 or 67.3 per cent, gross hematuria was the first symptom. This is slightly higher than the incidence of gross hematuria, as the initial symptom, in 902 cases reported by the Committee on Carcinoma Registry. The percentage in these cases was 63.5.

The order of occurrence of initial symptoms which directed these patients to seek medical advice are listed as follows:

Initial Symptoms	No. of Cases
Gross hematuria	142
Frequency of urination	40
Pain	9
Dysuria	7
Complete retention	3
Pyuria	2
Burning	2
Incontinence	1
Uncertain	5
Total	211

This group of cases consisted of 210 malignant tumors of epithelial origin and one sarcoma. Of the epithelial tumors in which bopsy studies were made thirty-nine were of the flat infiltrating type and 103 were papillary carcinomas.

Accurate history taking will elicit significant points regarding the patients occupation, since it has been shown by many investigators that among anilin dye-workers the incidence of cystitis, papillomatosis and papillary carcinoma of the bladder is much higher than in the ordinary population. None of the patients in our series gave a history of having worked in any of the dye industries.

Most tumors of the bladder occur between the ages of forty-five and seventy years and men are found to be afflicted about three times as often as women. In the group of cases we reviewed, 59.9 per

cent of the patients were found to be between the ages of fifty and seventy. The possibility of bladder tumors being incident to bilharzial infestation could be established by learning whether or not the patient had ever resided in a tropical country.

Physical Examination. Unlike the hypertensive, the peptic ulcer, or the gallbladder patient, the individual with cancer of the bladder is not a characteristic type. He may be of any physical stature and personality pattern. Careful physical examinations are prerequisites because the surgeon is then better fortified to plan his program of therapy. Secondary complications of cancer of the bladder should be searched for because they are frequently present. Rectal and vaginal examinations in cases in which there is palpable induration of the bladder wall are of great diagnostic significance for extension of cancer beyond the bladder wall may thus be detected.

Bimanual palpation is an important aid particularly in ascertaining the presence of peritoneal involvement, especially in tumors located in the bladder vault. Physical examination is usually negative in cases with pedunculated growths except when they are of extremely large size and in such instances they may be palpable suprapubically.

In presenting a complete picture of cancer of the bladder it is not sufficient to make the diagnosis and grade its malignancy, but every effort should be made to determine whether or not local or distant metastasis has occurred. This is very important because this one factor will frequently decide whether or not radical surgical treatment should be advised.

It has been rather generally accepted that metastasis from carcinoma of the bladder does not occur until late in the course of the disease. This is undoubtedly true for grades I or II but with the infiltrating tumors hidden metastases are probably present earlier than we suspect. In the report of the Tumor Registry the incidence of metastasis is 10 per cent. This figure, however, is based on physical and *x*-ray examinations and no estimate of the deep lymphatic or small visceral metastases could be included. The report also stated that from the data sent in on many of the cases it was evident that they had not been examined for metastases. A more diligent routine search will probably give us a higher percentage for metastases present at the time the patient is first seen. Such a study should include *x*-ray examinations of the lungs, lumbar spine and pelvis as well as a careful examination of the superficial and deep regional lymph-nodes and the liver. From the seventy-two cases on which sufficient information concerning metastases was submitted to the Carcinoma Registry the distribution was given in fifty-eight. These reports indicated that the bones were most frequently involved. Then in decreasing order of their frequency were the following: the lungs, regional nodes, liver, retroperitoneal nodes and the peritoneum.

Vault tumors with the rich lymphatic drainage along the hypogastric chain of lymphatics metastasize to the liver or by local extension involve the peritoneum. In one of our cases a vault tumor extending to the anterior bladder wall metastasized to the brain and the patient died with a hemiplegia. For practically all bladder carcinomas, except the tumors of the upper portion of the bladder, the first line of metastasis is along the lymphatic chains from the base and lateral walls of the bladder up to the nodes at the bifurcation of the aorta. The diagnosis of this extension is very difficult since these nodes almost never enlarge enough to produce edema of the lower extremities as is seen so frequently with embryonal carcinoma of the testicle and occasionally with carcinoma of the prostate. Metastasis to the lymphatics in the pelvis and along the lumbar spine may be suggested by pain in the lower part of the back, lower extremities and perineum. This is a referred pain and is due to involvement of the perineural as

well as the periovascular lymphatics. As this type of infiltration advances the bones of the pelvis, lower vertebrae and femures are invaded. When segmental resection, total cystectomy or ureteroenterostomy are contemplated, added significance is attached to the diagnosis of perivesical extension and metastasis, for in their presence these attempts to cure the patient completely would be futile. Since involvement of the deep pelvic lymphatics, the aortic nodes or those at bifurcation of the iliac vessels can be detected only by intraperitoneal exploration, this should always precede the attack on the bladder proper or the ureters. By opening the peritoneum one can also better judge the extent of the bladder tumor itself as well as peritoneal encroachment. This exploration can then be continued by palpation of the liver for the discovery of small, secondary nodules.

In the general evaluation of a patient with a diagnosis of cancer of the bladder his age, ordinary life expectancy, degree of present suffering renal function and infection and his ability to stand surgery must be considered before the appropriate treatment selected for his grade of tumor can be planned. While most infiltrating carcinomas are seen in people in the fifties and sixties, occasionally a grade IV, in the absence of demonstrable metastasis total cystectomy with large bowel transplantation of the ureters might be recommended. Yet a diagnosis of even a lower grade tumor in a poor risk or older person might call for cystectomy and cutaneous ureterostomy. With the same tumor diagnosis and a still worse surgical risk deviation of the urinary stream by cutaneous ureterostomy without cystectomy may afford some relief. The same relief is not obtained by suprapubic cystectomy because the trauma of the tube in the bladder again gives rise to pain. The indications for total cystectomy with ureteroenterostomy as well as the operative technic have been very clearly described by Priestley and by Higgins. Cutaneous ureterostomy with or without total cystectomy for carcinoma of the bladder has been advocated by Beer, Folsom and O'Brien and by Goldstein.

Urinalysis. Blood cells in the urine are found in all cases of cancer of the bladder. The blood may be present in various amounts from a few cells seen on microscopic examination to massive hematuria with many blood clots. Careful study sometimes reveals the presence of tumor cells. Pus cells are present in direct proportion to the degree of secondary infection. A small amount of blood in the urine is best detected by the orthotolidine test which is sensitive up to one part of blood in 24,000 parts of urine. The importance of blood in the urine even in minute amounts cannot be too strongly emphasized, and warrants complete urological investigation to determine its origin. It has been estimated that approximately 50 per cent of all patients with gross hematuria have bladder cancer. As mentioned above the incidence of gross hematuria as the presenting symptom in our group of cases was 67.3 per cent.

Classification. At one time the diagnosis of cancer of the bladder or papilloma of the bladder was enough information for a referring doctor. Depending on which one of these diagnoses was made, he was able to form an opinion regarding the probable course of the disease and the accepted treatment in either case. Now, with the advent of total cystectomy, the more frequent use of partial resection of the bladder, the question of how much fulguration and diathermy will do, the variable results of irradiation therapy and the problem of the proper disposition of the ureters, a more precise diagnosis is highly desirable. The type of tumor and the degree of its extension as well as its primary location and size determine what manner of management will best suit each individual patient. In order that the doctor who first sees a case of tumor of the bladder may give good counsel to his patient he should have a fairly good impression of what the urologist sees through the cystoscope and what the pathologist sees microscopically. To state that there is a papillary or sessile

growth would be inadequate information and to describe the tumor in detail would be confusing to those unfamiliar with cystoscopic pictures and histologic changes. To simplify the conveyance of the urologist's opinion a standard classification or grading of tumors of the bladder seems necessary. A generally used rough grouping is to refer to them as papilloma, papillary carcinoma or flat infiltrating carcinoma. A more accurate description would also take into consideration the character of the cells and the stroma architecture as well as the probable extent of infiltration into the bladder wall. Such a method of grading was advanced by A. C. Broders of the Mayo Clinic in 1922, when he classified epitheliomas of the genitourinary organs and placed them in four groups according to the degree of cell differentiation. According to this method all the epithelial tumors would be divided as follows: If about three-fourths of the epithelioma consists of differentiated epithelium and one-fourth undifferentiated, it is graded I; if the differentiated and undifferentiated epithelium is about equal, the tumor is graded II; if the undifferentiated epithelium forms about three-fourths of the tumor, it is graded III; if there is no tendency to cell differentiation, the tumor is graded IV.

The writers employ Broders' classification of bladder tumors and likewise call all epithelial tumors carcinomas. The so-called benign papilloma falls into the group referred to by Broders as grade I and this type of tumor is both grossly and microscopically benign. Cystoscopically, these tumors are observed to be composed of a short stalk or pedicle which springs from the bladder mucosa and ends in numerous frail branches or tendrils of various lengths which float in the fluid and are stirred by the least agitation or current. Microscopically the stalk is composed of a central blood supply surrounded by loose connective tissue and covered by layers of epithelial cells. Such a tumor presents no evidence of malignancy, either gross or microscopic at the time of examination but it is generally agreed that these are prone to undergo malignant change. These tumors may occur in various sizes and may occur as multiple growths in various parts of the bladder.

We have noticed a definite distinction between the single and multiple type of papilloma. The single so-called benign papilloma nearly always responds to electrocoagulation with a permanent cure. It is true that early multiple papillomas on histological examination are placed in the benign group; but because of their marked tendency to continue to recur over a period of many years, despite intensive treatment, the writers regard multiplicity as an evidence of malignancy and believe they should be classified as grade II.

Among the cases we reviewed there was one of a woman age fifty years who on first examination showed multiple papillary tumors. The bladder was opened suprapubically, biopsy specimens taken and the tumors destroyed by diathermy. In this process the entire bladder mucosa was seared as described by Deming. The histopathological report was multiple benign papillomas. This patient returned three months later to show on cystoscopic examination an extensive recurrence of papillary growths which on histological examination were definitely malignant and were classified as grade II. Total cystectomy was then performed.

Another case in this group demonstrates the long period of time over which these multiple tumors continue to recur. This patient was a male aged fifty-six, who was first seen at the age of thirty-nine complaining of gross, painless hematuria, and nocturnal and diurnal frequency of urination. About three years prior to his first visit he had one episode of gross, transient, painless hematuria. Cystoscopic examination revealed multiple papillary tumors over the base of the bladder. Suprapubic cystotomy with excision of the masses and cauterization of their bases with the Percy cautery was performed. Histological diagnosis was papillary carcinoma. He made

an uneventful recovery and was not heard from again for sixteen years. At that time he again complained of the same symptoms which had been present for two weeks. During the interim he had one attack of gross bleeding four years previously. On opening the bladder numerous papillomatous growths were widely distributed over the entire floor and vault of the bladder. Destruction of the tumors was accomplished with the Percy cautery. The specimen for biopsy showed papillary carcinoma grade II. He returned again at the age of fifty-six almost twenty-one years after his first attack of gross hematuria and almost eighteen years after the first destruction of his tumors. Suprapubic cystotomy was now performed for the third time and three superficially ulcerated growths, each the size of a pigeon's egg, with broad bases were removed. The pathologist reported them as grade II papillary carcinomas. However, we believe that a more correct classification for multiple tumors of this size with broad bases would be grade III.

Tumors that are classified as grade II present microscopic evidence of malignant change such as a tendency to produce numerous new branched papillomas with less piling up of the hyperplastic epithelium. Then, too, the microscope will reveal that the epithelial cells are hyperchromatic and their nuclei are of large size and irregular shape. A few mitotic figures may be seen. Cystoscopically, it may be impossible to tell a grade I from a grade II. Infection and ulceration of a papilloma are more prone to occur in a grade II than a grade I. Increase in the thickness of the pedicle as observed cystoscopically is an indication of malignancy and removes a papilloma from the grade I class to the grade II.

Tumors that fall into grades III and IV are of the infiltrating type and on microscopic examination the following features are noted: A small percentage of cell differentiation or absence of cell differentiation, numerous mitotic figures, squamous metaplasia, keratinization and pearl formation. The difference between a grade III and a grade IV cancer is one of degree rather than kind. The malignant features of a grade III cancer are present in a grade IV to a greater degree. Cystoscopic examination in such cases will reveal the papillary tumors to have thick pedicles and the mucous membrane at the point of attachment is thickened and adherent to the submucous structures. In general the villi are shorter and less regular in size and shape. Various degrees of necrotic change may be seen and the villi are very friable. The sessile types are quite nodular in character and in many instances an area of ulceration will be seen in the center of such a cancer. The bladder capacity is invariably reduced in cases in which the tumor is of a grade III or IV. Early sessile carcinomas may present very little cystoscopic evidence of their presence.

Cystoscopy and Biopsy. Cystoscopy is the most important procedure in determining the type of lesion causing hematuria. It will serve to differentiate the various urinary lesions which may cause hematuria, dysuria, frequency of urination, etc. Many workers rely almost entirely on the cystoscopic appearance for selecting the choice of treatment rather than the histopathological picture as disclosed in biopsy studies. We, however, believe that cystoscopy alone does not always fortify the surgeon with sufficient preoperative information. In addition to cystoscopy, biopsy and roentgen studies should be made by all means except when the tumor is obviously a grade I papillary carcinoma. The cystoscopic appearance of the epithelial tumors has been described above. Careful records of the cystoscopic findings should be made and they should include the size, number and location of the tumors. These records will prove valuable in surgical management and particularly for the periodic postoperative cystoscopic examination.

The ureters should be catheterized and specimens of urine from each kidney

examined for pus cells. If present, a Gram stain and culture should be made to identify the causative organism so that we may more intelligently manage the eradication of any upper urinary tract infection before surgical treatment is started. At this time the function of each kidney can be estimated and retrograde pyeloureterograms made.

Difficulties in cystoscopic examination are most frequently encountered in those cases of bladder neoplasia with marked secondary infection. In such cases infection should be combated carefully and the patient re-examined at a later date. The arrest of the infectious process will facilitate any subsequent surgical procedures. Incidentally, in clearing up these infections implanted on ulcerating carcinomas with the appropriate sulfonamide drug we have noticed a striking symptomatic improvement.

Hemorrhage will not prevent an accurate cystoscopic inspection except in extreme cases if care is taken to wash out all of the blood clots. The cystoscopic examination should be a very thorough one and if necessary, it should be done under anesthesia because a fleeting glance of a tumor is not adequate. The size, location, number, character of the pedicle, the type of tumor and evidence of infiltration are important points that one must ascertain by cystoscopic inspection. Tumors of grades III and IV are prone to undergo necrotic change and promote various degrees of secondary infection. Sessile tumors present crater-like ulceration in their centers, while papillary tumors may undergo severe sloughing of the villi and pedicle itself. The location of tumors has great significance since those on or about the trigone are not so amenable to excision or segmental resections as those occupying the movable portions of the bladder. It is unfortunate that the vast majority of tumors arise in the more fixed portion of the bladder. Some of these tumors are located so close to a ureteral orifice that their destruction or removal entails the sacrifice of the intramural portion of that ureter. In such cases intracystic transplantation of the ureter must be planned for as part of the operative management.

In a report of the Bladder Tumor Registry on a series of 902 cases of epithelial tumors their locations were listed as follows:

Region	Per Cent
Trigone	32.2
Lateral walls	35.3
Bladder neck	9.0
Posterior wall	10.2
Vault	7.5
Anterior wall	5.7

Over fifty cases of primary carcinoma of the bladder arising in vesical diverticuli have been reported. Higgins collected forty-nine cases from the literature in 1936; Heslin and Milner added one case in 1938 and Clancy reported another in 1941. Hematuria is again the cardinal symptom and was found in 77 per cent of the cases reviewed by LeComte, and 83 per cent of those reviewed by Higgins. On cystoscopic examination in cases with hematuria in which a vesical diverticulum is the only pathological finding, careful search for cancer concealed within the diverticulum must be made. Stones within the diverticulum may be easily ruled out by plain *x*-ray plate. In some cases the cystoscope can be introduced into the diverticulum and the cancer tissue observed.

Cystography is very valuable since filling defects will usually be seen in diverticuli that harbor a neoplasm. In some cases the tumor can be seen on cystoscopic examination through the mouth of the diverticulum or protruding from it. The authors have had experience with one case of primary cancer of the bladder occurring in a diverticulum and in this case cystoscopic examination was negative except for the presence of a diverticulum. The diverticular os was too small to permit insertion of the cystoscope; however, by gradually deflating the bladder the tumor plainly presented itself at the os of the diverticulum. We recommend this procedure as an additional aid in the cystoscopic diagnosis

of vesical carcinoma primarily located within a diverticulum.

The accuracy of biopsy diagnosis has been pointed out by Aschner who found that in 97.5 per cent of 242 cases the diagnosis was confirmed. Biopsy specimens are readily obtained by one of the various cystoscopic rongeurs. In securing these specimens care must be taken to include sections from the pedicle and base and with papillary growths very often most of the tumor has to be resected before the base can be exposed. The removal of the villous portion of these tumors by drawing the loop of the resectoscope into the sheath without the use of any current has the advantage of protecting the base and pedicle from the distorting effects of heat on the biopsy specimen. Occasionally, one can detect nodularity of the mucosa surrounding the pedicle. This is suspicious of extension and this area should have microscopic study. In taking fragments from the papillary growths it is important to select the more atypically shaped and more rigid fronds. A more composite histologic picture is achieved when different regions of the mass are represented. When the neoplasm is flat, ulcerated or necrotic, the central portion should be avoided since the margin of the ulcer with part of the indurated edge is more apt to show the true character of the lesion.

In certain cases in which submucosal invasion has progressed some distance from the primary site the cystoscopist will detect an abnormal adherence or fixation of the vesical mucosa to the submucosal structures. With grades III or IV tumors, sessile or papillary, we recommend the taking of several specimens from scattered areas in the bladder in an effort to determine the extent of suspected infiltration. This information is especially necessary when making a decision between segmental resection and total cystectomy.

Needless to say, the removal of specimens, from a thin bladder wall, ulcerated flat carcinoma or a tropic ulcer, requires considerable cystoscopic finesse to prevent the necessarily sharp jaws of the rongeur from including too much of the musculature in their bite. If there is the slightest doubt regarding the integrity of the vesical wall at the completion of the procedure, an indwelling urethral catheter should be kept in place for four or five days, and the patient should be closely watched for any signs of bladder perforation. If these are found, of course, suprapubic cystostomy and generous perivesical drainage especially at the site of rupture should be provided.

Improvements in the cutting current and the more popular use of the resectoscope have brought about a splendid advance in the treatment of tumors of the bladder but accurate diagnosis has probably been retarded by using the tissue, seared by the cutting current, for biopsy material. It is certainly true that when a large piece of tissue is removed in this manner the portion, most distant from the cut surface, will be practically just as valuable to the pathologist as if cut by the rongeur; but for our most important sections, when seeking early infiltration in a pedicle base or beyond the site of the visible tumor, the amount of tissue removed must, of course, be small. In these small sections the heat from the loop can alter the appearance so much that the pathologist will have difficulty in making a reliable diagnosis.

Another form of therapeutic progress which might easily lead to a considerable reduction in the percentage of completely correct diagnoses is electrocoagulation. The refinements in electrocoagulating equipment and the excellent results they so often produce may very well tempt an optimistic urologist to destroy the tumor without taking a biopsy specimen. The obvious danger in such a practice is that occasionally on cystoscopic examination a tumor appears small and innocent enough to respond to electrocoagulation but actually has a high percentage of undifferentiated cells and unsuspected invasive powers. After one or more recurrences with attempts at destruction by heat a histo-

pathological diagnosis is desired. At this stage, however, the biopsy material will often consist of a section of sloughing surface leaving undisturbed the deeper malignant process or the secondary inflammation may make a diagnosis still uncertain. In this manner one can see how such diagnostic management can repeatedly delay the institution of proper treatment. A more satisfactory method which avoids these pitfalls in the diagnostic process is to remove bits of tissue from precisely selected areas of the tumor or bladder and then use electrocoagulation when this is indicated by the cystoscopic appearance. Then after histological examination, if a higher grade of malignancy is found, we can proceed with more radical treatment.

Cystography and Urography. Valuable information can be obtained if a series of cystograms are made in cases suspected of being infiltrating cancers. Reference is made particularly to those cases in which extensive infiltration of the bladder wall is present and cystoscopically one may see a relatively small and innocent looking lesion. Cystograms or pneumocystograms of such cases will show irregularities in the mural contour of the bladder, the site of infiltration being represented by an area in which the bladder wall does not stretch normally. Such findings are in our opinion almost pathognomonic of an infiltrating neoplasm. With invasion of the intramural portion of a ureter we look for cystographic evidence of a reflux of the contrast medium in the affected ureter. The cystogram may also be useful in determining the size and location of certain papillary tumors but it should be employed essentially as a supplement to the cystoscopic examination. Careful and complete studies of the upper urinary tract should be made because the type of surgery, planned for handling cancer of the bladder, may depend on the status of the kidneys and ureters. In some cases ureteral obstruction will be present as a result of invasion of the intramural segment of the ureter. Under such circumstances dilatation of the ureter and various degrees of hydronephrosis and pyelonephritis may have occurred. Occasionally, the obstruction of the intramural segment of the ureter will enable one to indict as infiltrating a tumor which appears cystoscopically to be of a lower grade. Furthermore, if such a tumor is located some distance from the orifice of the obstructed ureter one may assume that it must be a very malignant growth if it has traveled, hidden beneath the mucosa, for that distance. Briefly it may be said that the greater the distance between the primary neoplasm and the obstructed ureter the greater the degree of malignancy.

Complete knowledge of the condition of the ureters is again imperative when the surgeon elects to divert the urinary stream. Obstruction and ureteritis may have produced dilatation and areas of stenosis. With ureters which are very much dilated ureteroenterostomy is difficult and hazardous, while on the other hand such ureters are well adapted for cutaneous ureterostomy. Cutaneous ureterostomy with small calibered ureters leads to considerable discomfort occasioned by the large tubes necessarily kept continuously in the ureters to the renal pelves. If smaller tubes are used, they are apt to provide inadequate drainage and require too frequent replacement.

Differential Diagnosis. In the differential diagnosis of carcinoma of the bladder one must consider several other prominent causes of gross hematuria as well as a few of the causes of dysuria, frequency and urinary retention. The hematuria and other symptoms of acute or subacute cystitis will frequently lead one to consider tumor of the bladder. In some of these cases even on cystoscopic examination extensive bullous edema, ulceration, grey exudate or thickening of the bladder mucosa may appear the same as that seen about a carcinoma. When there is no underlying pathological lesion in the bladder or adjacent structures, the inflammatory

process will readily respond to the appropriate urinary antiseptic. When the cystitis is secondary to carcinoma of the bladder, there is often a similar subsidence of symptoms and a disappearance of the edema, exudate and mucosal congestion while the patient is having local therapy or taking the urinary antiseptic. However, this improvement quickly gives way to an exacerbation of the cystitis, on discontinuing treatment, if a tumor is present. This treatment of the superimposed infectious process can be of great diagnostic assistance. Usually about one week after therapy is started the second cystoscopic examination will reveal an unmasked tumor which can then be clearly inspected. In the meantime this process will prove very gratifying to the patient for in the absence of or even with a decrease in the severity of the inflammatory reaction he is appreciably relieved of his dull continuous pain, dysuria, frequency and urgency and even retention of urine and hematuria may be less pronounced.

The removal of the disguising infection is especially important prior to securing material for histologic examination since biopsy in the presence of severe cystitis is unreliable and may be dangerous. The specimen removed may include only the edematous portion and leave intact the deeper malignant cells. When inflammation alone is present, the cutting blades of the rongeur can bite so much of the bladder wall that the infection can spread perivesically and set up retroperitoneal infection or peritonitis.

An incrusted cystitis can resemble carcinoma so closely that only by biopsy can they be differentiated. Its chronicity and the ammoniacal urine with blood, pus, mucus and necrotic tissue suggest carcinoma. Then on cystoscopy, the calcareous incrustations and necrotic membranes, often forming pseudofronds, can still cause uncertainty regarding malignancy, since the flat infiltrating carcinoma may present an identical appearance. The finding of urea-splitting organisms in the urine is of little help because they also commonly accompany carcinoma.

Cystitis cystica can occasionally resemble carcinoma of the bladder. The characteristic, small translucent cysts of cystitis cystica may be so closely grouped that they seem confluent. At this stage the collection of cysts can appear exactly like a patch of bullous edema and for this reason carcinoma may be suspected. The lack of induration in the bladder wall and biopsy will make the diagnosis certain.

Cystitis follicularis with its solid, rounded, nodules of lymphoid tissue beneath the epithelium is not likely to be confused with carcinoma unless the urologist is searching for extension in the vesical wall from an infiltrating carcinoma elsewhere in the bladder. Biopsy may be necessary for final diagnosis.

Renal, ureteral or vesical calculus can simulate tumor of the bladder by the gross hematuria and the frequency they produce. A stone impacted in the intramural portion of the ureter can seldom lead one to think of a tumor. With stone in the upper urinary tract the renal colic or dull pain indicate that there is also ureteral obstruction. With urograms and catheterization of the ureters the presence or absence of stone is easily determined. Only on rare occasions is this type of pain an early symptom in carcinoma of the bladder. However, in the later stages the tumor nearly always invades the region of one or both ureteral orifices and causes obstruction. The infection and renal damage incident to this type of obstruction from infiltration of carcinoma account for more deaths than those caused by metastatic lesions or perivesical extension of the disease. Caulk, in analyzing the records of the cause of death in ninety cases, found renal disease in 38 per cent of them. Of these cases twenty-five came to autopsy and in fifteen or 60 per cent the cause was in the kidneys.

Acute diffuse nephritis is not an uncommon cause of hematuria and it frequently must be distinguished from surgical conditions in the urinary tract. Here urinalysis

will show more albumin than one would expect from the amount of blood in the urine and hyaline and granular casts will be found if a centrifuged specimen is examined. The symptoms and findings in nephritis can easily delay the diagnosis of an early co-existent cancer of the bladder. Only by cystoscopy or urography can we be entirely certain about such a complication.

Benign prostatic enlargement, carcinoma of the prostate and varicosities in the posterior urethra are causes of gross hematuria, dysuria and frequency, but by combinding the findings of rectal examination and cystourethroscopic examination there will be little difficulty unless an infiltrating carcinoma of the base has invaded the prostate as it does in about 12 per cent of bladder carcinomas. Carcinoma of the prostate may also extend upward at the bladder neck. In either case biopsy is often necessary to establish the site of origin.

Tuberculosis in the bladder has symptoms similar to those of cancer of the bladder but in very few cases will cystoscopy and visualization of the upper urinary tract fail to distinguish them. The characteristic destructive process in the kidney and the chronic inflammatory changes in the ureter are distinctive. The ulcers of tuberculosis are usually multiple and have little elevation in contrast to the ulcer of the single sessile carcinoma.

Hunner's ulcer (localized submucous fibrosis) in its more severe form can possibly be confused with carcinoma. The symptoms of severe pain, frequency and intermittent hematuria together with ulceration in a contracted bladder, which is difficult to inspect without general anesthesia, are somewhat similar to those of a flat infiltrating tumor. A long history of frequency and pain which is knife-like in character and intense when the bladder is full and made worse by jarring the body is typical of Hunner's ulcer. The diagnosis is made of cystoscopy which will show ulceration or linear abrasions of the mucosa which bleed easily when touched or on distention of the bladder. They are practically always on the vault or moveable portion of the lateral and posterior walls.

Extravesical inflammatory lesions such as diverticulitis, carcinoma of the rectosigmoid, acute appendicitis, pelvic inflammatory disease, dermoid cyst and endometriosis can cause a localized reaction with bullous edema and congestion of the bladder mucosa. A grey exudate, ulceration and bleeding frequently are seen. The localized lesion is usually on the posterior or lateral walls. The cystogram may give a picture very similar to that of infiltrating carcinoma with rigidity in part of the bladder wall. Vaginal or rectal examination and bimanual palpation will usually disclose the nature of the extravesical lesion. Sigmoidography combined with the cystogram is informative especially if there is an intestinal diverticulitis or carcinoma of the sigmoid. When an endometrial implant invades the bladder it appears on cystoscopic examination as a tumor usually between the ureteral orifices or just posterior to this location. If it has involved the mucosa, it bleeds easily and is associated with bullous edema. A history of aggravation of the chief symptoms, which are frequency, dysuria and suprapubic pain, during the menstrual periods prompts a further search for signs of endometriosis in other locations such as a nodular thickening in the cul-de-sac. Cystoscopic biopsy, on an endometrial tumor in the bladder and on the other extravesical lesions, should be done only when all other efforts have failed to establish the diagnosis. The danger of biopsy in these conditions is that it may aid in the formation of a vesicointestinal fistula.

Trophic ulcer, the result of irradiation of the cervix or body of the uterus, may be easily mistaken for a flat, ulcerated carcinoma of the bladder base. The cystoscopic pictures may be identical and the symptoms of frequency, hematuria and dysuria, which is intense, are so protracted and disabling that cancer is always suspected. The microscopic examination may mislead the pathologist and he also may make the

diagnosis of carcinoma of the bladder because of the late tissue changes caused by irradiation. The ulcer nearly always occurs in the midline just behind the interureteric ridge. It is produced by the blood supply to that part of the bladder wall. It would be a very grave error to consider this lesion a carcinoma and for it advise more radiation therapy, which obviously would lead to disasterous results.

On cystoscopic examination the ulceration is seen in sizes varying between 5 mm. and 5 cm. in diameter. It is accompanied by bullous edema and a chronic inflammatory reaction around it while in the center is a grey slough or necrotic area.

Vaginal examination is very enlightening because the thickened indurated bladder floor of carcinoma is not felt even with very large trophic ulcers.

The history of having had irradiation of the uterus will aid in establishing the diagnosis but since the ulceration occurs between one and ten years after exposure the patient may fail to volunteer this information. In studying the time of onset of bladder symptoms in twenty-four patients Dean found that the average was two and one-half years after irradiation.

Bilharziasis of the bladder may easily be confounded with carcinoma if we rely on the cystoscopic picture alone. The early lesions are small papules with raised, yellow centers or ulcers. Later, sessile or papillary granulomas develop and these have a neoplastic appearance. With ulceration there is secondary infection with calcareous deposition and induration of the bladder wall. Biopsy will distinguish it from carcinoma and in the biopsy material the ova may be seen. The urine always contains these ova and they can usually be demonstrated. They appear as long ovids with narrowing at one end where a spine is attached. Bilharzial infestation in the terminal venules and capillaries of the submucosa is followed by deposits of ova which become encysted and it has been noted that in these areas of the bladder malignancy itself not infrequently develops. The history of having visited Africa or Egypt is a significant aid in diagnosis.

Mesothelial tumors of the bladder may be rebenign or malignant and in either form a very rare. They are distinguished from carcinoma by biopsy since there is little difference in their cystoscopic or cystographic appearances. The rapid growth and failure to produce symptoms until the mass is very large suggest that it is not an epithelial tumor. The epithelium seems to remain intact and hematuria is a late symptom. One of our cases was a sarcoma in a man forty-two years of age. Hematuria was the first symptom and was not noticed until two weeks before admission to the hospital when we found a spherical mass, completely filling the bladder, attached by a small pedicle. The more firm benign mesothelial tumors are frequently found by the patient who palpates the mass in the hypogastrium.

Secondary carcinoma from a papillary carcinoma of the renal pelvis may be seen protruding from a ureteral orifice or as multiple papillary growths distributed closely about one ureteral orifice. These are eliminated from diagnostic consideration as primary bladder tumors by demonstrating the primary renal carcinoma and possible ureteral growths by retrograde pyeloureterograms.

Recurrent carcinoma of the bladder at the site of a previously destroyed or removed tumor may present difficulties in diagnosing its presence or in differentiating it from a new, primary carcinoma arising elsewhere in the bladder. A detailed record of the location of the original growth will distinguish between the new tumors and recurrences. However, changes brought about by irradiation or electrocoagulation used in the treatment of the original tumor may hide the early recurrent carcinoma. The microscopic as well as the cystoscopic appearance may be deceiving. An increasing area of ulceration, persistent bleeding and marked calcareous incrustation should

make one suspect a recurrent or remaining carcinoma. Usually within six months after the destruction of even a large tumor the healing process is complete. It is only by familiarity with the ordinary tissue response to heat and irradiation and by repeated cystoscopic inspection that we can recognize the new tumor tissue, springing up in the midst of a devitalized area or in the surrounding zone of inflammation, early enough to apply effectively the same treatment or to institute hopefully more radical measures. It is for this reason and also to discover new growths early that the urologist should keep an additional appointment book just for patients with tumors of the bladder. The ordinary words of caution and instruction to return on a certain date do not seem impressive enough to bring the symptomless patient back for re-examination. Routine letters reminding them of the importance of keeping this condition under control and assigning them specific dates for examination may prove more persuasive. Counseller and Walters request these patients to return twice at three-month intervals, then six months later and again in one year. These examinations should include periodic cystoscopy regardless of the absence of microscopic hematuria, for new tumors or a true recurrence may attain a large size before the patient is examined at a time when red blood cells appear in the urine.

SUMMARY

The important clinical and particularly the diagnostic features of carcinoma of the bladder have been examined. The value of the case history, physical examination, urinalysis, classification of epithelial tumors, cystoscopy, biopsy, roentgen studies and differential diagnosis have been outlined. Certain points have been emphasized by reference to cases which the writers have examined. The writers believe that the problem of cancer of the bladder can be handled in a more satisfactory manner if the surgeon is fortified with complete knowledge of the urinary tract prior to operation.

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* Due to lack of space a few references have been taken out. However, these references will be included in the reprints.

General

DIAGNOSIS OF DISEASES OF THE THYROID GLAND

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WE believe that diseases of the thyroid gland amenable to surgical attack are always associated with enlargement of the gland, in whole or in part. The first question to be answered, therefore, in examining a patient suspected of having a disorder of thyroid *function*, is "Has he a goiter?" If the answer is in the negative, the overwhelming probability is that clear evidence of thyrotoxicosis will not be brought forth by systemic examination and by laboratory investigations. The writer regards as a myth the view that there is in the human subject any degree of thyroid intoxication not accompanied by enlargement of the gland. We see, not infrequently, thyrotoxic patients whose goiters seem obvious to us and in whom the diagnosis of toxic goiter is confirmed anatomically but in whom enlargement of the gland is not detected by able physicians though they have readily recognized the systemic manifestations of hyperthyroidism. We see rather more patients diagnosed elsewhere as being hyperthyroid in whom we can neither confirm the diagnosis of hyperthyroidism nor detect any enlargement of the gland. Without wishing to labor the point one may add that there is no substitute for experience in the palpation of enlargements of the thyroid and we have not, personally, seen a case in which the diagnosis of hyperthyroidism was borne out by anatomical diagnosis or by clinical response to nonoperative management in which the enlargement of the gland was not palpable. (It should be understood that this statement bars from consideration goiters which are entirely intrathoracic.) There is ample opportunity for humility, however, when one attempts to predict the exact size or extent of the goiter and the precise nature of the goitrous tissue, as will be discussed. Suffice it to say that it is axiomatic with us that the goiter will prove to be bigger at operation than we have anticipated from examination of the neck.

EXAMINATION OF THE NECK

In a favorable light the diagnosis of even small goiters can be made with considerable certainty at a distance by observation of the rise and fall during deglutition of the shadow when the skin is molded by the pressure of an enlarged thyroid. Asymmetry of an obvious swelling will ordinarily indicate the presence of a nodular gland. The presence of rapid expansile neck pulsations transmitted from the carotid arteries and jugular veins gives an indication of the cardiac rate and is, further, an expression of the wide pulse pressure of thyrotoxicosis. Similarly, flushing of the skin of the neck is due to arteriolar and capillary dilatation and is common in thyrotoxicosis though by no means pathognomonic, particularly if transient, for it accompanies the rapid fluctuations of arterial blood pressure and vasomotor tone seen in patients with a variety of conditions characterized by emotional lability. Congestion of the external and anterior jugular veins and a dull suffusion of the face, sometimes unilateral, indicate such obstruction of the tributaries of the superior vena cava as occurs with goiters of considerable size which fill the superior thoracic strait or descend into the anterior

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mediastinum. Further examination will indicate whether this phenomenon is a concomitant or is independent of congestive heart failure; the two situations frequently co-exist. Wildly, irregularly filling superficial jugulars are often a clue to auricular fibrillation. Auscultation over the cardinal vessels (superior and inferior thyroid arteries) will reveal the presence or absence of bruits. These are *rarely* heard in the absence of hyperthyroidism. Light palpation over the vessels enables one to detect thrills in the arteries; again, practically pathognomonic. Thrills and bruits are less apt to be present in the lower grades of hyperthyroidism, especially those associated with nodular goiters, and they disappear under treatment somewhat earlier than the precordial, blowing systolic murmur so characteristic of rather acute hyperthyroidism.

The position of the patient during palpation of the neck may help or hinder accuracy in examination. The patient is ordinarily examined in the sitting posture and instinctively extends the cervical spine, raising the chin and putting the sternomastoids and prethyroid muscles on such tension as to make more difficult palpation of the gland. The cervical spine, therefore, should be in an easy, slightly flexed position during preliminary palpation in order to relax all the muscles superficial to the gland. The lateral lobes and isthmus should be felt for in an effort to describe their size, surface characteristics, consistency and mobility on the trachea. The height to which the upper poles extend toward the angle of the jaw and the degree to which lower poles extend toward or beneath the clavicle can best be brought out by having the patient swallow while the examining fingers are gently pressing on the portion of the lobe in question. Details of one lobe can be brought out best then by having the neck rotated *toward* the lobe in question in order to relax the sternomastoid on that side. The writer prefers to stand facing the patient, generally, and to examine the patient's right lobe with his left hand and the left lobe with his right hand. In each case, the effort is made to palpate the lobe between the thumb, lying medial to the sternomastoid, and the index and third fingers lying lateral to it; meanwhile, the fingers of the other hand, by gently pressing backward on the opposite lobe, rotate the lobe in question *forward* between the examining fingers and thumb. This maneuver is particularly useful in demonstrating the smaller of two lobes in an asymmetrical goiter. At times, further details can be added by standing behind the patient and carrying out palpation of each lobe with the thumb behind the sternomastoid and the index and third fingers anterior to it. Finally, in cases in which the presence of enlargement of the gland is very doubtful or the question of fixation on the trachea is in question, additional information may be obtained by examining the patient in the supine position, since the tumor lies higher in the neck in this position.

The position of the trachea is always of interest and may be of vital importance. It is ordinarily in the midline of the neck in symmetrical, bilateral goiter and may, with a small isthmus, be palpated throughout its extent between the lobes. But if covered anteriorly by an enlarged isthmus and a pyramidal lobe or by a rotated unilateral enlargement it may not be palpable at all. Fetal adenomas of considerable size, particularly when solitary, produce the most marked deviations of the trachea from the midline and their presence should always be suspected when such deviation is found. We are always interested in the character of the opposite lobe in such a case for in the Mississippi Valley we are used to seeing fetal adenomas in glands which are *otherwise* abnormal, i.e., the remainder of that lobe and the opposite lobe usually show varying grades of colloid goiter or of diffuse hyperplasia (particularly the former) or of mixtures of all three processes. On the contrary, authors in the Southwest say that they usually find the solitary adenoma in an otherwise normal

gland. Enucleation of the adenoma is obviously adequate in the latter situation, whereas subtotal thyroidectomy is more suitable to the former in which there is no normal thyroid tissue grossly detectable at operation. The writer must own to a definite percentage of error in the preoperative estimation of the character of the opposite lobe in such cases. The error always lies in the direction of assuming that the opposite lobe will be normal in those cases in which it cannot be palpated with certainty before opening the neck. We are used to finding considerable degrees of *rotation* of the trachea in such cases and in them the opposite lobe may be easily twice normal size and grossly nodular without being felt in the examination of the intact neck. Varying degrees of rotation and tipping of the larynx itself is often present in these cases, particularly if the adenoma is quite large and lies high in the neck; and it may be exceedingly difficult to intubate such patients if intratracheal anesthesia is chosen for them. Retrotracheal extension and enlargement of the opposite lobe is clearly indicated in the case in which one lobe is obviously enlarged, regardless of its character, yet the other lobe is not definitely felt, *if the trachea is in the midline.* In such cases it is the retrotracheal extension of the opposite lobe which *prevents* displacement of the trachea away from the palpable lobe.

The retrotracheal projections of, generally, nodular goiters and the retrotracheal extension of the upper poles, particularly, of diffuse goiters cannot be made out with any certainty in most cases in the intact neck and this is all the more reason that they should be sought out at operation by delivery of the gland from its bed before excision is started; such portions, if overlooked, are, in our own experience, the most common cause of persistent hyperthyroidism. We have come to expect to find deep lying, often retrotracheal upper poles in patients with short, thick necks and the author has not noted elsewhere in the literature in explanation of it the rather obvious fact that such patients have, as part of their general physical structure, *short superior thyroid arteries* which, particularly if they lie anterior to the thyroid tissue at the upper pole, limit its enlargement in the anterior direction. This seems to the writer a more plausible explanation than the usual one of strong, thick prethyroid muscles in these individuals of sthenic habitus who have also a wide aorta, wide costal angle, short mesentery and correspondingly short abdominal vessels. This observation may well have been made previously but the author has not encountered it and it is certainly not a part of general knowledge of this aspect of the subject.

There are numerous symptoms and physical signs that suggest the extension of goiter into the upper thoracic cage. Typical is the patient's statement that he formerly had a large goiter which has greatly diminished in size or entirely disappeared. The writer has, at the moment, such a patient in the hospital who gave such a history preoperatively and only the very top of whose left lobe could be felt in the neck; it rose but little on swallowing, yet the patient could *cough* the mass (the size of an orange) up into the neck through the superior thoracic strait. Such intrathoracic masses are nearly always extensions downward of goiters whose upper portions can be felt more or less easily in the neck, yet in rare instances an adenoma descends into the thorax and its connections with the remainder of the lobe in which it originated become so attenuated as to be unrecognizable at operation. The author has seen only one such case personally, a thyrotoxic individual with a bilateral diffuse hyperplastic goiter whose lateral lobes were some three times normal size and had small retroclavicular extension of the lower poles. The usual rather radical subtotal thyroidectomy was followed by considerable improvement but with unmistakable persistent hyperthyroidism. Only after eighteen months did a small mass become palpable in the suprasternal notch

and a completely intrathoracic mass the size of a grapefruit was then discovered and removed. At the second operation the remnants of the lateral lobes could barely be identified and there were no fibrous or vascular connections between the structures in the neck and the intrathoracic mass. Complete relief of hyperthyroidism followed the second operation and the anticipated hypothyroidism developed within six weeks. Normal thyroid status is, of course, maintained by thyroid feeding.

A spasmodic, nonproductive cough, episodes of near-suffocation and decided dyspnea on exertion (especially in the absence of hyperthyroidism) are suggestive of tracheal irritation and partial obstruction commonly found in patients with intrathoracic extensions of goiter. The brassy cough, occasional hoarseness and "crowing," particularly on exertion and during sleep, strongly suggest pressure or traction on the inferior laryngeal nerve, but we find these symptoms and signs to be present far more commonly without evidence of nerve injury than with such evidence from visualization of the larynx. The occurrence of venous engorgement characteristic of obstruction of the upper vena cava and its tributaries in connection with intrathoracic goiter has been mentioned previously. Dullness on percussion of the clavicle and subclavicular areas can readily be demonstrated also. Absence of breath sounds anteriorly over the apex of the lung is often found on auscultation. Finally, intrathoracic goiter must be ruled out in those rare patients in whom strong systemic evidence of hyperthyroidism is present with a wholly "unsatisfactory" amount of enlargement of the lateral lobes to account for the systemic findings. In our experience such extensions have occurred almost entirely in individuals of sthenic or hypersthenic habitus. In them the normal location of the gland is not only closer to the superior thoracic strait than that of the asthenic person but the strait itself is larger. It is a fact in such persons that one sees usually nearly as large a portion of big goiters below the clavicles as above them.

Palpation of the neck in suspected persistent or recurrent hyperthyroidism offers a wholly different set of circumstances for physical examination due to the manner in which scarring in the plane of the pretracheal fascia may limit the forward extension of the enlargement of gland tissue. The diagnosis of thyrotoxicosis is made in any case on systemic, rather than on local, findings and, *provided* the original operator followed the practice of dissecting out the upper poles completely, persistent or recurrent hyperthyroidism in its early stages is usually due to overlooked retrotracheal or substernal projections which continue to hyperfunction and to enlarge in the depths of the neck where they may not be palpable until relatively late.

The methods of examination accessory to the use of one's God-given special senses in the study of the goiter itself are the laryngoscopic mirror and the roentgen ray. All patients for whom thyroidectomy is contemplated should have an inspection of the larynx in order to detect the occasional unsuspected inferior laryngeal nerve paralysis, to determine on which side the involvement occurs, to rule out perforation of the larynx or trachea by suspected carcinoma of the thyroid and to exclude intrinsic diseases of the larynx itself. We regard the existence of inferior laryngeal nerve paralysis as being a practically absolute indication for a two-stage thyroidectomy (regardless of the presence or absence of hyperthyroidism), the lobectomy on the affected side being performed *first* and the second lobectomy deferred until such time as to allow ample opportunity for maximum recovery of function of the impaired nerve. Operative injury to the uninvolved nerve previous to such recovery precipitates, of course, the tragedy of double abducens paralysis and this is a situation that can be tragic enough even though it be but temporary. Finally, only by routine postoperative visualization of the larynx can one honestly evaluate his

own incidence of nerve injuries and thereby alter, as necessary, his technical procedures when the incidence of this complication, frequently unsuspected if unilateral, gets beyond the inevitable minimum experienced by all of us.

X-ray examination of the upper chest and lower neck in the anterioposterior plane and, as necessary, in oblique planes, together with lateral views of the neck are most useful in locating exactly the position of the trachea throughout its course and for the more or less exact definition of retrotracheal and intrathoracic extensions of goiter which are often calcified. We depend on them to guide us toward a more complete evaluation of the mechanical problems involved in certain cases. Whenever it is financially feasible for the patient they make an unquestionably desirable addition to the *routine* of examination of all goiter patients. That they are not routinely feasible in all private practice (i.e., when the patient's own money, rather than someone else's money is being spent on them) is equally unarguable. Only the good judgment of the physician can decide when they are indispensable in the individual case. In any case, it is often possible to achieve the ideal of a routine *x*-ray film at small cost to the patient by the exercise of ingenuity in obtaining roentgenograms in combination with other specialized forms of expert service from the same source, if the physician is not prepared to deliver them from his own resources. Hospitals are showing a desirable tendency to insist on routine studies in their clinical laboratories on all patients admitted and to set a "flat rate" fee for such admission examinations for a decidedly smaller sum than would have been charged for such examinations if ordered as optional examinations in previous years. Yet the writer suspects that more widely useful information would be obtained from the inclusion of an *x*-ray film of the chest in such a routine than, let us say, the usual estimation of the nonprotein nitrogen concentration of the blood. One is even more confident that the accuracy of the film would be higher than that of the biochemical determinations done in the average hospital laboratory.

Occasionally, signs are found on palpation which seriously raise the question of the presence of the relatively uncommon disorders of the gland not considered heretofore. These special findings are hardness of the tissue, fixation of the gland on the trachea or to the overlying muscles and marked tenderness. Acute thyroiditis is prone to follow acute upper respiratory infections and actually occurs far less frequently as a consequence of hematogenous infection during septicemia than one would reasonably anticipate. In our own limited experience it has nearly always occurred in a pre-existing goiter, generally adenomatous, rather than in a presumably normal thyroid gland and, in the days before chemotherapy with the sulfonamides, went on to suppuration requiring surgical drainage more often than it resolved. The involved portion of the gland is swollen, very tender and painful and usually becomes fixed by inflammatory reaction to the surrounding tissues. There is frequently local heat, edema and redness of the overlying skin. Fluctuation is not usually demonstrable until very late because of the tenseness of the capsule and the edema of the prethyroid tissues. One should not wait for demonstrable fluctuation before draining these abscesses.

Hardness and fixation of the gland are common to both chronic thyroiditis of the Riedel type and to carcinomas of the thyroid which have perforated the capsule. Constriction of the trachea is produced in the former case by the contraction of scar tissue; invasion of the trachea is commonly the earliest extension of carcinoma beyond the capsule. Both conditions are accompanied by extension of their peculiar processes to the prethyroid muscles; but regional lymph gland metastasis is frequent in carcinoma whereas enlargement of regional nodes is not characteristic of Riedel's struma. Both diseases are fre-

quently associated with adenomatous goiter and the great majority of carcinomas of the thyroid arise in adenomas. This leads, of course, to the special consideration of adenomas of the thyroid as being always potential "dynamite" as far as the risk of the appearance of carcinoma of the thyroid is concerned. While the fact that approximately 5 per cent of fetal adenomas show carcinomatous degeneration on removal does not mean that 5 per cent of all fetal adenomas undergo malignant degeneration; yet one can only regard such an argument in favor of watchful waiting on adenomas in adults as being wholly specious when one considers that perforation of the capsule and fixation of the gland by invasion of surrounding tissues is the *first* sign that indicates the presence of carcinoma. No one who has watched a number of deaths from carcinoma of the thyroid will condone an expectant policy with adenomatous goiter, regardless of the state of thyroid function. That true adenomas occur both in glands otherwise normal and in glands otherwise diseased is again beside the point. Certain types of tumors which must be grouped as carcinoma of the thyroid show blood vessel invasion before fixation to adjacent fibrous structures occurs and in them the prognosis is necessarily guarded even though the local excision of the growth is complete.

We have been impressed with the aching discomfort experienced by some of our own patients with Riedel's struma and by the occasional finding of tenderness to palpation in the earlier cases. These are of infrequent occurrence in carcinoma of the thyroid in its early invasive stage before nerve involvement has taken place. It should be noted particularly, however, that the occurrence of sudden pain and increase in size of a previously recognized nodule and the presence of tenderness, all of which are classical signs of hemorrhage into an adenoma, may mark instead the perforation of the capsule of the adenoma by carcinoma with no associated hemorrhage whatever. We have recently seen such a case at the onset of the discomfort; operation performed within a week of the event disclosed the findings outlined. Finally, the microscopic differentiation between Riedel's struma and certain carcinomas may be exceedingly difficult and the final answer may be obtained in such cases only by the clinical course of the patient.

There are no certain gross criteria for the recognition of Hashimoto's disease (struma lymphomatosa). The process does not extend beyond the capsule of the gland and occurs almost exclusively in smooth goiters. It is associated with normal or decreased thyroid function and is of rare occurrence.

THE RECOGNITION OF HYPERTHYROIDISM

The estimation of abnormal thyroid function, let it be repeated, does not depend upon the examination of the neck but upon recognition of the systemic effects of increased or decreased thyroid activity. This is not to say that we may not find evidence on examination of the neck that the goiter, if present, is toxic in all likelihood, as has been outlined in the previous discussion. Yet the local signs which, on examination of the goiter itself, suggest toxicity are almost entirely circulatory phenomena, thrills and bruits over the superior and inferior thyroid arteries, expansile pulsation of the neck and abnormalities of the filling of the superficial jugulars. These may be equivocal and some of them, particularly the carotid and jugular phenomena, are not specific for thyrotoxicosis. They are, finally, characteristic only of acute or refractory hyperthyroidism of a fairly high grade and may not be present at all in relative remissions of the functional disorder. The generalization is a fair one, therefore, that one should make the effort to estimate thyroid function first without reference to neck findings, then to correlate the two sets of data. The more comprehensive is one's experience with thyrotoxicosis, the more rarely will appear apparent paradoxes between local and systemic findings.

The diagnosis of hypothyroidism and myxedema are outside the scope of this discussion since they are not, in themselves, indications for thyroidectomy as is, by and large, hyperthyroidism. While we are not concerned here with the indications for thyroidectomy, it may clarify our dismissal of the subject of hypothyroidism from further discussion to say that only certain types of goiters (Riedel's struma, questionable carcinoma, adenomatous and the large colloid goiters) seen with hypothyroidism or with normal gland function may properly be considered operable; whereas the goiter associated with thyrotoxicosis is nearly always properly to be considered as a surgical problem at one time or another regardless of its pathological classification. More simply, in decreased or normal function of the gland the pathological classification of the goiter or associated mechanical and cosmetic features constitute the indications for or against thyroidectomy; in thyrotoxicosis, the abnormal function itself constitutes the indication.

Thus we come to the problems involved in the recognition of thyrotoxicosis. In no field of medicine has more gratuitous nonsense been preached by individuals unfamiliar at first hand with the course of the patient throughout the disease in its varying development and progress. Thyrotoxicosis does not differ from many other disorders in requiring a fairly concentrated experience with its manifestations, the opportunity for prolonged observation of patients, preferably in more than one generation of the family of these individuals, and leisure for careful consideration of the rapidly shifting phenomena of the disease before the thoughtful observer will have developed a mature and comprehensive view of it. Many of the factors militating against a clearer understanding of the disease are to be blamed on the faulty observations and misconceptions of a previous generation and these are perpetuated as myths in current thinking on the subject; others are being introduced by members of the writer's generation in medicine, usually in the form of ill digested bits of experimental efforts whose results (!) have been translated into terms of clinical medicine by observers profoundly unfamiliar with thyrotoxic patients. There is much that is obscure in the origins of hyperthyroidism and in its disturbances of intermediate metabolism, but the confusion and ignorance among physicians of the phenomena that characterize the clinical course of the disease are largely the result of poor clinical and didactic teaching in our medical schools and the number of articles accepted for publication by editors who are apparently unable to evaluate them critically. Thyrotoxicosis in its clinical manifestations is not nearly so mysterious as we have made it.

An intelligent evaluation[1] of the thyroid status of the patient suspected of having hyperthyroidism can only be made with regularity if the examiner is familiar with the *spontaneous course* of the disease and with the degree to which it may also have been altered by treatment previous to his examination. Fundamentally, the spontaneous course of hyperthyroidism, it cannot be said often enough, is by remissions and relapses. Such a cyclic course is most pronounced in the patients with the diffuse hyperplastic goiter most often associated with exophthalmos, somewhat less striking in the patient with multinodular toxic goiter and hardly noticeable in the patient with the solitary, toxic adenoma. In addition to this conditioning of the severity of relapses and of recovery toward a baseline of health (normal thyroid functional status) in the remissions by the *pathological type* of the goiter, there is an important modification of the cyclic characteristic with the passage of time, for the gradual downhill course over prolonged periods, in terms of years, tends in general to produce a chronic, refractory type of hyperthyroidism at the more severe levels of the disease, with only transient and minor phases of improvement. Whether the essentially cyclic nature of the disease is due to a recurring stimulus whose intensity varies

or whether it consists of a cyclic response by the gland to a constant stimulus (if, as seems likely, stimulation from a source outside the thyroid is essential to the development and maintenance of hyperthyroidism) is beyond the scope of this discussion. One may, therefore, count on the occurrence of fairly complete remissions, *given time*, early in the course of the disease but, on the other hand, should expect only relatively minor improvement in the patient much farther along the road regardless of how long one waits. We take advantage of these facts in deciding when to perform thyroidectomy in the individual case and in choosing between one-stage and two-stage thyroidectomy.

A most striking illustration of this is the patient of the type, seen occasionally by every surgeon of considerable experience in this field, who will be intensely thyrotoxic for a period of several weeks after coming under treatment, in whom operation is quite properly deferred and who returns after several months with not merely a return to normal thyroid status but with myxedema. It was the observation of such cases that led us to abandon preliminary ligation[2] of the superior thyroid arteries as being not only dangerous but of no therapeutic merit, in 1931. We learned to expect quite as rapid (or as slow) improvement in patients deferred over the same periods of time without ligation and obviated the mortality of 7 per cent which ligation carried in our hands between 1925 and 1931. We have records of patients so deferred whose basal metabolic rates have fallen below zero after a lapse of months of continuous treatment. One frequently sees, of course, the patient at a time when his thyroid function is normal, as judged by all tests not involving the performance of work by the patient, but who has been clearly shown to have been moderately thyrotoxic at a previous date by a thoroughly competent observer. A carefully taken history, then, will trace the development of the disease and will mark the occurrence of such episodes previous to the time of examination and will, taken in conjunction with physical and laboratory data, permit one to chart the patient's position with considerable accuracy on the individual course his disorder has taken. One is also enabled to predict with fair accuracy the probable future course of the patient, if left to his own devices, but the physician who values his reputation as a prophet will bear in mind that the *duration* of remissions and relapses is uncertain. The writer is personally familiar with two obdurate individuals who were gravely informed by a surgeon of wide reputation during episodes of severe thyrotoxicosis over ten years ago that they would be dead within six months unless they consented to operation. Their precarious maintenance of chronically impaired health justifies them, in their own minds, as advertising their would-be benefactor as an alarmist and "radical."

The spontaneous course of thyrotoxicosis will be modified, *but not essentially altered*, by treatment in varying degrees as the ideal combination of rest, sedatives and iodine is, or is not, achieved. A detailed account of previous treatment is essential to the interpretation of the history and the current physical and laboratory findings. It is a permissible digression at this point to say that everyone with a professed opinion on hyperthyroidism should be familiar with the work of Means[3] and his co-workers on the relative effects of the continuous administration of iodine and its alternating administration and withdrawal in "courses" which is unfortunately still fashionable. Their publications are the standard work on the subject, in the writer's opinion, and bear out most graphically our own observations which have led us since 1930 to administer iodine continuously to thyrotoxic patients from the time of their first examination until some weeks after operation. We fully share Means' view that iodine does not itself induce refractoriness to its own effects but that the patient who is getting into a relapse in the more severe grades of

thyrotoxicosis is *relatively* more refractory then to iodine, as to all other measures, than he is at a time when he is starting on a spontaneous remission.

One approaches the hyperthyroid patient, then, with the view that the disease is cyclic in character and that the point at which the patient finds himself on his own course at a given time is conditioned not only by the type of goiter involved but by the duration of the disease and by such previous treatment as he may recently have had. It is not our purpose to detail here the signs and symptoms of thyrotoxicosis, but to recall a plan[1] for the recognition and evaluation of the acutely thyrotoxic individual and to point out the necessity for prompt institution of treatment for these individuals who are generally getting worse at the time of first examination (or they would not request it) and are often on the verge of crisis. The manifestations of thyrotoxicosis are somewhat erratic at times in that expression of the disease may be much more pronounced in one organ-system than in others and it is axiomatic with us that the patient may get worse (or improve) in different respects at different rates. The more *inclusive*, therefore, the system of assaying the signs of severity of the disease, the less apt is one to overlook a single important clue to impending crisis when all other signs are reasonably satisfactory. This is equally important in estimating the risk of operation at any given time and extremes of physiologic derangement in any of the respects outlined below constitute, in fact, *absolute* contraindication to operation at such a time. The validity of these views has been proclaimed by Willard Bartlett, Sr. for many years and the general trend to individualization[4] in treatment of thyrotoxic patients, current during the author's participation in surgical practice, has been importantly shaped by his efforts.

PRIMARY CRITERIA

1. *Circulatory.* The more extreme grades of tachycardia, with either normal cardiac mechanism or auricular fibrillation, are warning signs. The writer has seen only two instances in which cardiac decompensation has occurred with *regular* rhythm, one of them a woman of eighty-four with severe co-existent hypertension, the other a diabetic patient of sixty-five, also a hypertensive individual. Almost invariably, therefore, auricular fibrillation will appear before decompensation occurs and, in fibrillators, the *first sign* of a break in compensation is the appearance of a pulse deficit; its presence or absence must be definitely established in all fibrillators. Decompensation does not occur, in the belief of the writer, in thyrotoxic patients who have not another disease of the circulatory system and he has encountered but one case in a patient under forty years of age. In a series of decompensated patients seen over one five-year period, the average age of these individuals was forty-eight years in comparison with the average age of thirty-five years for all thyrotoxic patients admitted during the same time. Certainly, the circulatory apparatus of the person past fifty years withstands the added burden of hyperthyroidism much more poorly than does that of the younger person. By far the commonest co-existent circulatory disease in our own patients who become decompensated is hypertension. Moreover, the overwhelming majority of our patients admitted with decompensation are severely thyrotoxic and have become decompensated in an exacerbation of thyrotoxicosis. Their basal metabolic rates, determined after the restoration of compensation, have usually fallen between plus 40 and plus 65, which is high indeed for patients in this age group.

The existence of low concentration of protein in the blood plasma, often with a reversal of the albumin-globulin ratio, was described by Shirer[5] in 1932 and has been confirmed by Bartels,[6] Brown and McCray[7] and in unpublished data of the writer. It was attributed by Shirer to incomplete assimilation of ingested protein from the small intestine which is known to show

increased mobility and rapid emptying in thyrotoxicosis. Bartels attributes it also to reduced production of protein by the liver, the principle source of plasma protein. The writer[8] has postulated a loss from the blood due to capillary damage caused by the severe acidosis[9] recently shown to be characteristic of the disease. In any case with edema, therefore, as pointed out by Shirer (whose paper has come to the writer's attention only during the preparation of this article), decreased concentration of the proteins of the serum or plasma should be sought as another possible etiological agent beside myocardial insufficiency.

It is worth mentioning that no decision can be made with certainty as to the presence or absence of hypertension in patients who are first seen with decompensation until after thyroidectomy has been performed and normal thyroid balance regained. It is similarly true that the diagnosis of diabetes mellitus in the glycosuric patient with moderate grades of hyperglycemia cannot be proved or disproved in the presence of severe hyperthyroidism. But the existence of two diseases affecting the circulatory apparatus must always be regarded as the probability in the early evaluation of the decompensated patient with toxic goiter; and the complicating diseases, in order of their frequency in the writer's experience, are hypertensive heart disease, arteriosclerosis (out of proportion to the patient's age) without hypertension and valvular lesions of the heart. Decompensation constitutes an absolute contraindication to operation, as long as it persists.

2. *Nutritional Status.* The patient who is losing weight at a rapid rate in an acute phase of the disease may easily get into a precarious situation. One sees occasional cases in which a gain in weight to a new high level for the patient occurs at the onset of the disease. Such patients, to the best of the writer's knowledge, have not been studied critically. The obvious possibilities are two: their caloric intake is sufficiently in excess of their energy requirement that fat is actually stored or they are having gradual storage of water, presumably in association with depletion of plasma proteins, in clinically undetectable amounts.

The writer wishes to record here an observation that has intrigued him for years and of whose validity he is confident. This is a rather rapid gain in weight previous to the onset of symptoms of hyperthyroidism as being so frequent a finding in a carefully taken weight history as to be almost characteristic. While at this high weight level, briefly maintained, the symptoms of thyrotoxicosis occur and the traditionally observed loss of weight ensues. It is our belief that the original gain in weight above the patient's previous average is a part of the disease process that eventuates as thyrotoxicosis. We believe, furthermore, that statistical study will show that the incidence of obesity in persons who develop hyperthyroidism is importantly higher than that for the general population. Herz and Means[10] have commented on the onset of hyperthyroid symptoms following profound losses of weight, however induced, but they have not, apparently, been impressed with the occurrence in their patients either of a preceding rapid gain in weight or of the significance of obesity of long standing. As a matter of fact, weight losses should be reported and evaluated not only in terms of pounds or kilograms, but in *percentage of body weight.* Thus, we can say offhand that the patient who has lost over 25 per cent of body weight as a result of disease, regardless of its nature, is generally in a precarious situation and this is particularly true in hyperthyroidism. A large percentage of our two-stage thyroidectomies are performed on patients in whom such weight losses have occurred. We regard a weight loss at the rate of more than one-half pound per day under hospital treatment as being an absolute contraindication to operation.

3. *The Nervous System.* One is tempted to think that only lack of acquaintance at first hand with the general public could lead to the attribution of such various vagaries of behavior as have been blamed on thyrotoxicosis. Hyperthyroid individuals are not the only ones who behave eccentrically, have poor judgment and emotional instability and are restless and irritable. Thyrotoxicosis has even been discussed seriously as being itself a psychoneurosis and the onset of hyperthyroidism is frequently attributed to worry. The writer would cut through all such discussion by stating that intimate and prolonged familiarity with thyrotoxic patients, their parents and their children, convinces him that thyrotoxicosis merely accentuates the normal characteristics of the individual. In other words, hyperthyroidism makes "nervous" persons more "nervous." The not infrequent occurrence of thyrotoxicosis in a usually calm, emotionally mature individual does not turn that patient into a flighty, irresponsible person. The late appearance of such patients has been graphically described by Lahey[11] in his striking term of "apathetic" hyperthyroidism; but I believe that one underestimates Dr. Lahey's mastery of his subject if one takes his characterization to mean that behind the patient's "dead pan" face, overstimulation and visceral intoxication are not proceeding just as in less phlegmatic individuals. Certainly, in our view, the presence of emotional immaturity, outbursts and irresponsibility raises the serious probability that there are two disorders present, only one of which is hyperthyroidism, affecting the behavior of the patient. The proper time for exact differential diagnosis is *previous* to operation and thyroidectomy undertaken as a sort of therapeutic test of the patient's psychic status is apt to have shocking repercussions. If more exact differential diagnoses were carried out on these individuals, there would be fewer tales among laymen of patients who have "lost their minds" following thyroidectomy. In the thyrotoxic patient, the presence or recent occurrence of *acute thyrotoxic psychosis* is closely associated with thyroid crisis and constitutes an absolute contraindication to operation. The importance of cerebral edema in crisis has recently been pointed out by the writer.[12]

4. *Excretory Systems.* It is most uncommon to see abnormalities of renal function as a result of hyperthyroidism. But the gastrointestinal tract and the skin give important indications of the severity of the disease at times. Increased frequency of stool is rather characteristic of our own patients and the occurrence of vomiting, diarrhea or of four to six stools (even though formed) per day in a previously constipated individual may herald the approach of crisis. While it is inexact in terms of physiologic and biochemical derangements, it is clinically useful to think of the brain, the circulatory apparatus and the gastrointestinal tract as all tending to show in varying degree evidence of stimulation by toxic goiter. When dissipation of body heat lags behind its production under conditions of moderate environmental temperatures, gross sweating occurs as the insensible perspiration[13] increases with rising metabolism. The author, as surgical house officer under Dr. Coller, used to hear him say that thyrotoxic patients never got pneumonia. This is a very fair generalization, barring, of course, the terminal bronchopneumonias of crisis. We recall having seen but one postoperative pneumonia; it was associated with lobular atelectasis and aspiration of ingested water associated with injury of the inferior laryngeal nerve. The presence of vomiting, diarrhea and excessive sweating are absolute contraindications to operation.

5. *The Basal Metabolic Rate.* The level of basal metabolism in thyrotoxicosis is always of interest to us. Its use and limitations in differential diagnosis will be discussed later. In serious grades of hyperthyroidism its tendency to rise, to remain stationary, or to fall during periods of close observation is of more consequence than

the actual levels through which its variations are measured. A *rising* metabolism is an absolute contraindication to operation.

6. *Duration of Voluntary Apnea (D.V.A.): the Breathholding Test.* This test was first applied to thyrotoxic patients and its usefulness perceived by Willard Bartlett, Sr., in 1924, and the writer has had the opportunity of developing it,[14] modifying the technic to its present form, standardizing the interpretation of the results[15] and finding in acidosis the probable biochemical explanation[8] for its behavior. No detailed consideration of the technic of the test will be given here. We regard it as being the most useful *single* method of estimating the severity of thyrotoxicosis in a given situation, for any of the more advanced signs of acuity, as elaborated in the preceding paragraphs, are reflected in the ability to hold the breath. Its *inclusiveness* is of the utmost value to us, taken in conjunction with other clinical and laboratory data, in deciding when operation is safe at all and whether a one-stage or two-stage thyroidectomy will be done.

Of the various laboratory procedures other than the estimation of basal metabolic rate, only mention of the determination of plasma proteins (already alluded to) and of the various tests of liver function will be made, since their proper sphere of usefulness is not in the diagnosis of hyperthyroidism, but in its treatment. Blood cholesterol and amylase will be referred to later.

DIFFERENTIAL DIAGNOSIS OF THYROTOXICOSIS

There is ordinarily little difficulty in the recognition of acute or severe hyperthyroidism if the characteristics of its more serious stages, as set forth above, are part of one's organized knowledge. The text books classically list pulmonary tuberculosis, cancer and diabetes as being the diseases from which it must be differentiated, yet cachexia, common to the advanced phases in neglected cases of all of these disorders, is the only common denominator among them. One must be aware that there is no law against the thyrotoxic patient also having another disease. We see the co-existence of toxic goiter and, variously, diabetes, carcinoma (especially of the female breast), psychoneuroses and hypertensive heart disease not infrequently, as noted previously, and one will always have to be prepared for such findings in examining patients who have reached middle age. Yet it is a tribute to the success of the concerted attack on tuberculosis in the past two decades that the writer has not seen (or has not recognized) the co-existence of thyrotoxicosis and active pulmonary tuberculosis in a single instance in private practice. Let us leave the discussion of the situation in which the patient has two diseases, one of them a toxic goiter, only with the warning that it "can happen," that a complete diagnosis should be made before thyroidectomy is performed and that the actual existence of diabetes or hypertension in the thyrotoxic patient may sometimes be demonstrated with *certainty* only after restoration of normal thyroid status has been achieved, however strong the previous suspicion of their presence.

The eye signs of hyperthyroidism have not previously been discussed because of the growing conviction of the writer that they are almost irrelevant to the diagnosis. This statement wants qualification: (1) If the examiner does not use an exophthalmometer, the simplest instrument being that devised by Luedde,[16] he is able to recognize exophthalmos only of the most severe grades and is unable to measure the considerable variations of individual patients that occur during their clinical course; (2) measurable grades of exophthalmos may be seen with the microscopic picture of hyperplasia in the thyroid regardless of the various gross pathological characteristics of the gland (diffuse or nodular enlargement); (3) it is the present impression of the writer, based on unpublished data, that lid signs are more con-

stantly associated with disorders of thyroid function than are measurable grades of exophthalmos; (4) severe exophthalmos is often associated with hypothyroidism, rather than with hyperthyroidism, and is reversible, in the early phases, by thyroid feeding;[17] (5) the writer believes that exophthalmos, lid-lag and extra-orbital muscle palsies in patients with disorders of thyroid function will eventually be proved to be due to wholly indirect effects of the latter. The limitations of space preclude any consideration of the evidence for these statements. The fact is, practically, that positive eye signs are not *alone* pathognomic of thyrotoxicosis and may be present with normal thyroid function or with hypothyroidism. That they are apt to be present in diffuse hyperplastic goiter with thyrotoxicosis is unarguable.

The commonest confusion in the minds of physicians in general arises in the differential diagnosis of questionable thyrotoxicosis from (1) psychoneurotic states and (2) states involving pluriglandular deficiencies. In both of these situations the patient with a goiter is in serious danger of thyroidectomy, though the goiter may be only an innocent bystander. Where no such complicating conditions are present, the recognition of the thyrotoxic patient, if seen first *during a remission*, offers problems of its own. Some of the characteristics of remissions, spontaneous or induced, have already been considered in the discussion of the spontaneous course of thyrotoxicosis. If the patient has recently taken iodine, the decision as to whether the patient has previously been hyperthyroid, and is now in a remission, or has never been thyrotoxic at all, can be puzzling indeed. Obviously, if the patient has in fact been toxic, the remission is the ideal time to perform thyroidectomy and one will be secure in the knowledge that important subjective benefit will ensue. On the other hand, if the symptoms, however alleviated during brief treatment (iodine or otherwise), are due to some disorder other than hyperthyroidism, the patient stands an excellent chance of being made worse by operation. It is too much to hope that the status quo will be maintained after any major operation.

Allusion has been made repeatedly to the fact that considerable or complete remission may be expected with the passage of time, regardless of treatment, in the *early* cycles of thyrotoxicosis, particularly when the goiter is not nodular. In such cases, all objective findings made under resting conditions, including basal metabolic rates, may be well within normal limits which are elastic at best. We have proposed an original method for recognition of such patients and we are confident that *the response of the circulation to measured exertion* is of great value in the clarification of this situation. The writer has recorded several hundred such tests and finds quite characteristic patterns[18] in the results.

Technique. Under resting (not necessarily basal) conditions, the pulse rate and pulse pressure are estimated repeatedly until no further diminution in the activity of the circulation occurs. These are recorded as "resting" values. The patient then hops twenty times on each foot in a period of about twenty-five seconds and resumes the sitting posture. The pulse rate and pulse pressure are then determined immediately and at intervals of one minute thereafter for the first five minutes of the recovery phase.

One sees three characteristic responses in the patients under consideration: (1) Patients with nontoxic goiter, whether their thyroid function is normal or subnormal, respond by a moderate, immediate increase in both pulse rate and pulse pressure with recovery to resting values within two, or at most three, minutes. (2) Hyperthyroid patients in remission (even though the basal metabolism is below zero, as in numerous cases of the writer) show an acceleration of pulse rate or a very large increase in pulse pressure, or both, immediately after exertion and a *failure to return to resting levels* in one or both phases (especially the pulse pressure) within three

minutes. (3) Patients whose thyroid function has not been abnormal, but who have psychoneuroses (especially the anxiety states) characterized by circulatory symptoms, show *little or no increase of pulse pressure* in response to exertion and it may actually fall below resting levels immediately after exertion or during recovery; pulse rate has always returned to resting values within three minutes in these patients.

Such objective evidence as to thyroid function from the response to work is of great value, taken in conjunction with subjective data (to be discussed) in trying to decide whether the goitrous patient has a low grade hyperthyroidism in remission, or has normal thyroid function (or hypothyroidism) plus a "living problem" which has eventuated in a neurosis that more or less closely simulates hyperthyroidism. The regular performance of the exercise test decreases considerably the number of therapeutic tests with iodine otherwise necessary and permits one to carry out such therapeutic tests with at least a more enlightened attitude. If therapeutic tests are to be done, their value is greatly enhanced (in that positive or negative results can be freed from the taint of suggestive therapy) if *serial* estimations of plasma cholesterol and amylase concentrations can also be obtained. The principal deviation of cholesterol (elevation) is most regularly observed in hypothyroidism and the decided usefulness of knowledge of its behavior in this situation has been emphasized by Hurxthal.[19] The variations of plasma amylase are complementary, its greatest deviation from normal (depression) in hyperthyroidism having been originally described[20] by the writer. Single estimations of either of these substances are not of great use in the differential diagnosis of the particular problem with which we are presently concerned, their ranges of normal values being considerable, yet their shifts, even though these be within the ranges of normal, in response to specific therapy, are most suggestive in the evaluation of thyroid function by therapeutic tests. They have the merit of not being subject to the numerous upsetting influences that are reflected in the level of "basal" metabolic rate.

It is to be hoped that the reader will not assume from the immediately preceding paragraphs that in the case of the patient with an apparently innocent goiter and complaints roughly simulating hyperthyroidism, inexplicable on any basis of organic disease, it is necessary always to carry out the methods of objective examination detailed in addition to the usual routine of careful, comprehensive physical and laboratory study before arriving at a definite conclusion. On the contrary, these accessory methods are not usually necessary but they will give information of decided value in those occasional cases in which the skillful physician finds himself most skeptical of the validity of his own conclusions. The performance of such tests is in itself a measure that will often win the respect and confidence of a patient whose oustanding characteristic is loss of self-confidence and who has already been examined by the usual methods by others with inconclusive results. Certainly, the additional understanding of these basic deviations of physiologic and biochemical characteristics engenders in the physician a confidence in the direction in which he is going that is easily communicated to the patient who may have been distressed by attitudes of bland reserve previously encountered.

No group of patients exists whose problem is more challenging to one interested in man's combat with his environment than that composed of the individuals who are currently classed as being "functional" cases. This term is probably to be taken as evidence of our present inability to measure the organic terms that constitute inferiority of physical make-up, the microscopic or physicochemical inadequacies that result in the scale being tipped toward a breaking down of the sense of well-being under the stress of problems of living. Much can be

learned from an organized analysis of what the symptoms mean to the patient, even though the physician has not the intuitive gift of sensing frustration, fear and hopelessness in the person so afflicted. The possession of what Hugh Cabot has termed "a low threshold of suspicion" is a priceless asset if one is to recognize such characteristics in the patient, realize the *validity* of symptoms arising from problems inseparable from the business of living and translate them into terms of treatment intelligible to one who is bewildered enough when he comes to the physician for help. The analysis of such subjective symptoms has been detailed elsewhere[18] and their differentiation from the symptoms of hyperthyroidism, which they may more or less closely simulate, is not ordinarily difficult. Such an evaluation of symptoms should precede physical and laboratory examinations. It will not be obtained, except by good luck, hurriedly, or by the inexperienced, or by "hired hands." Inquiry should be made into the following ten points though the patient may not volunteer information about some of them while being very voluble about others:

1. *Nervousness.* Pronounced emotional coloring and frequent presence of phobias characterize the neurotic patient's complaint. The effect of such feelings is to alter the pattern of behavior much more than in the case of the thyrotoxic individual, who tends to keep on at his usual tasks, whereas the neurotic typically withdraws more and more from participation in the ordinary routine of living.

2. *Heart Consciousness.* A variety of expressions of concern over the behavior of the circulatory apparatus is exemplified by the frequent complaint that the heart cannot be felt to beat at all, or that it is going to stop altogether; this may alternate with a sensation of overactivity, of palpitation or of precordial pain. The association with exacerbations of emotional stress is more noticeable than with exertion, distraction of attention and relief often being obtained by even purposeless activity.

3. *Loss of Weight.* The coincidence with either loss of appetite or discomfort after eating small amounts is constant; the common result is diminished food intake. We have seen more extreme grades of emaciation with anorexia nervosa than with thyrotoxicosis.

4. *Shortness of Breath.* This is to be sharply differentiated from dyspnea on exertion though this latter may also be present. It consists of sighing respiration, occasional overventilation with tetany, or a "smothering" sensation relieved by changing position and, especially, by access to moving air.

5. *Neck Sensations.* These are uncommon in toxic goiter or in other goiters in which decided evidence of interference with the airway is lacking. A sense of constriction, "tightness" in the neck and choking sensation, especially on excitement, are typical.

6. *Weakness.* This usually is present on arising in the morning and is accentuated by the contemplation of a task, rather than by its accomplishment. It leads to avoidance, not only of physical work, but also of social contacts and the effort implied therein.

7. *Inability to Sleep.* Difficulty in getting to sleep associated with worrying in bed and light sleeping are common, as is the feeling of wakening completely unrefreshed. All statements as to the duration of sleep are most unreliable.

8. *Abnormal Temperature Sensations.* While the thyrotoxic patient is notoriously intolerant of heat and the hypothyroid patient of cold, the neurotic individual is apt to be distressed by both extremes of temperature. Perspiration from cool extremities is characteristic, as is profuse axillary or facial sweating, particularly on excitement.

9. *Digestive Disorders.* A multiplicity of complaints is again common here. Flatulence, dyspepsia after a few mouthfuls, the most impressive consciousness of peristalsis and concern over the daily stool are observed. Various phases of "colitis" with

mucus in the stool are occasionally present. Extremes of diarrhea or of constipation and their accompanying phenomena may quite overshadow the circulatory or nervous complaints as the history is developed.

10. *Menstrual Disturbances.* While hyperthyroidism generally tends toward diminution in the amount of flow and may occasionally be accompanied by the suppression of the menses, the history of decided irregularity, periods of amenorrhea, excessive flow and late appearance of the menarche should prompt the suspicion that an endocrine imbalance may be responsible for at least some portion of the sympathetic instability of certain of these patients. Menopausal symptoms may also be confusing.

Above all, the impact of the personalities of these individuals should be one that enables the examining physician to put together the threads outlined in this brief sketch of the characteristics of their symptoms and to observe how they weave a pattern of interference with normal duties, withdrawal from the business of living into a constantly diminishing circle that yet has, typically, brief moments of a sense of well-being quite unlike the rhythm of hyperthyroid fluctuations. These latter are measured in weeks or months, rather than in hours or days. These unhappy patients, moreover, *complain* of their health. The thyrotoxic patient is so apt to be intoxicated and overstimulated that he is almost unaware of fatigue and tends to minimize even serious disability.

EVALUATION OF THE BASAL STATE

Finally, a brief consideration of the estimation of basal metabolic rate is appropriate for it is most difficult to determine accurately in the patients in whom it is most needed. The important errors, as far as the diagnosis of hyperthyroidism and its differential diagnosis are concerned, are not made at levels above, say, plus 20 per cent, but between the levels of zero and plus 20. The ideal state which we wish to induce at the time of the test is, after all, that obtained just after waking and before arising in the morning. It is fruitless to hope to reproduce such a state after 9 A.M. in a person who has been awake since 6 A.M., who has slept badly, who has assumed some of the household responsibilities before proceeding to the laboratory, who has been a lengthy trip through metropolitan traffic (especially in a crowded public conveyance) or who is burdened emotionally at the time of the test. The means whereby we strive to obviate such upsetting factors are not to our point here, which is to recognize by objective methods that we either have or have not achieved a truly basal state. This evaluation of the "basality," or lack of it, is all important if one is to interpret wisely the rate estimated from the oxygen consumption under those particular circumstances. If the physician does not have the data on which to review each test critically, the estimation of basal metabolic rate will be confusing or misleading about as often as it is helpful in evaluation of the thyroid status.

We have been gathering such data since 1938 and the plan to be outlined not only has merit but is the only one known to us which gives the desired information and does not force us to fall back on clinical judgment alone when the reported metabolic rate is at variance with our expectation. It consists merely of a comparison of the pulse rate and pulse pressure determined under (1) frankly nonbasal but *resting* conditions in the office with (2) similar data recorded, usually on the day immediately preceding or following, under allegedly basal conditions at the time of the estimation of metabolic rate. The circulation, as judged by the pulse rate and pulse pressure, should show measurable slowing under conditions purporting to be basal. Those metabolic rates obtained in patients with normal thyroid function, hypothyroidism, or hyperthyroidism which are most readily acceptable in view of clinical data have uniformly been accompanied by such decrease in both pulse rate and pulse

pressure. We have been fully confirmed in our rejection of such other tests in which significant diminution of pulse rate and pulse pressure did not occur by clinical data, other laboratory procedures and repetition of the tests. On several occasions on which we have rejected the metabolic rate as not being basal, we have found the pulse rate and pulse pressure to be practically the same under resting conditions after luncheon on the day of the test and have repeated the metabolism determination then and there, finding approximately the same oxygen consumption in the middle of the afternoon as under the allegedly basal conditions of the morning. We believe that during the test itself periodic recording of the pulse rate alone is sufficient to indicate the occurrence of discomfort, nervousness, or fatigue which has not been present at its beginning; and more than minor variations of pulse rate are, of course, sufficient to cause us to view the result with suspicion. We are satisfied that with these circulatory criteria we can at least evaluate the degree to which we have achieved, or have failed to achieve, an approximation of the basal state and to discount or to reject intelligently those reports of the metabolic rate obtained when considerable slowing of the circulation has not occurred. Such a routine requires patience and attention to detail, but does not differ in so doing from all other worth while methods, clinical or laboratory, which are useful in the evaluation of the thyroid status.

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DIAGNOSIS IN ACUTE PELVIC CONDITIONS

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DIFFERENTIAL diagnosis of acute pelvic conditions, as, indeed, all diagnosis, is dependent upon evaluation of minute details of the patient's own story and on performing an examination which is sufficient to detect or rule out the various probabilities.

At the expense of appearing trite, it seems not out of order to emphasize these requisites. The clinician who develops his own story from direct conversation with the patient, giving particular attention to the sequence of events and the manner in which the history is related, has an infinitely better chance of arriving at a correct diagnosis than he who depends on a history recorded by another person. It is extremely difficult, if not impossible, to convey the fine shades of difference of meaning which the same written words may imply. In this connection also should be mentioned the importance of giving full credence to the patient's story, at least, until it is proved to be false. More diagnoses are missed by discounting some apparently minor statement or flatly refusing to accept the patient's story than by the clinician's being deliberately misled. The author will not soon forget that as a student he missed the opportunity of diagnosing a spontaneously developing gastrocolic fistula because he refused to accept a Pullman porter's statement that fifteen minutes after eating whole cherries, he would pass the seeds by bowel.

The examination, both clinical and laboratory, must be comprehensive enough to include an opportunity to detect the likely cause of trouble. Even the relatively inexperienced clinician is able to interpret the findings of routine examinations. Most of our errors are attributable to failure to make certain examinations and tests rather than to our inability to interpret those which are performed. This point is well illustrated by the not infrequent error of mistaking a right basal pneumonia for acute appendicitis because of failure to examine the chest with this possibility in mind.

Because of the necessary limitation of this presentation it is impossible to discuss in detail every cause of pelvic pain in women. Only the more frequent conditions will be considered and only the most significant diagnostic points will be developed. The author disavows any claim of presenting new or original thoughts on this subject but it is hoped that a review of some of the well established diagnostic criteria may be of some benefit.

For the sake of clarity, the chief diagnostic points of each of the major causes of pelvic pain have been tabulated, and the table is followed by a brief discussion of each condition. It seems wholly unnecessary to state that no single sign or symptom in these lists is pathognomonic of any condition and that only by taking the clinical picture as a whole can one reach a sound decision.

ACUTE PELVIC INFLAMMATION

Chief Diagnostic Points

1. Follows some provocative cause:
   - (*a*) Menstruation
   - (*b*) Abortion
   - (*c*) Childbirth
   - (*d*) Instrumentation
   - (*e*) Excesses (sexual and alcoholic)
2. Bilateral pain
3. Tenderness greatest near Poupart's ligament
4. Fever relatively high (101 to 103°F.)
5. Leucocytes relatively high
6. Sedimentation rate very rapid

7. Evidence of infection in lower genital tract
8. Bilateral pelvic masses

In the absence of some provocative cause for its ascent, it is well known that gonococcal infection of the lower genital tract tends to remain below the level of the internal cervical os. Of such causes, menstruation is by far the most frequent as is evidenced by the fact that most patients with acute tubal inflammation date the acute onset near the end of a menstrual period. In most of the remainder, one or another of the listed provocative causes can be elicited.

Because of the wider area of involvement in gonococcal pelvic inflammatory disease, the fever tends to be high, the leucocyte count high and the sedimentation rate rapid as compared to these findings in acute appendicitis. The bilateral distribution of pain, if present, renders differentiation relatively easier.

The patient who presents the above picture should be examined for evidences of gonococcal infection of the lower genital tract, giving particular attention to Skene's and Bartholin's glands as well as the cervix. As it is almost, if not quite impossible to reach any decision relative to these structures with the patient in bed, the necessity of examining patients on a proper table with adequate light is quite evident.

While evidence of gonococcal infection of the lower genital tract is almost conclusive proof of pelvic inflammatory disease, one must remember that such evidence does not completely eliminate the existence of some other condition which may require immediate operative treatment.

Palpation of tender bilateral adnexal masses in a patient who presents a typical picture leaves no doubt of the existence of pelvic inflammatory disease. In many of the very early cases, however, the inflammatory process is still in the catarrhal stage and in these the only positive findings may be bilateral adnexal tenderness, and the production of sharply increased pain on lateral motion of the cervix.

ACUTE APPENDICITIS

Chief Diagnostic Points

1. Occurs unrelated to periods
2. Often provoked by intestinal upset
3. Typical sequence of symptoms:
   (*a*) Generalized pain
   (*b*) Localization of pain
   (*c*) Nausea
   (*d*) Vomiting
4. Tenderness unilateral, localized at McBurney's point
5. Low fever (99 to 100°F.)
6. Relatively lower leucocyte count than in pelvic inflammatory disease
7. Relatively slower sedimentation rate

While the gynecologist readily admits that acute appendicitis is not always a pelvic condition, the frequency of its mistaken diagnosis in patients who do have pelvic lesions, leaves it in the controversial territory between the gynecologist and the general surgeon.

The history will reveal the classical sequence of the symptoms in most cases of appendicitis. Relationship to the usual provocative causes of pelvic inflammatory disease cannot usually be established. Less elevation of temperature and of the leucocyte count, the relatively slow sedimentation rate and typical localization of the pain and tenderness are invaluable points in differentiating appendicitis from inflammatory disease. The presence of fever and the classic sequence of symptoms along with an entirely negative pelvic examination point to appendicitis as opposed to hemorrhage from a ruptured Graafian follicle or corpus luteum.

OOPHORRHAGIA

Chief Diagnostic Points

1. Definite relationship to menstrual cycle
   (*a*) Midway—follicle
   (*b*) One week before—corpus luteum
2. Pain is low
3. Shock and severe hemorrhage are rare
4. Nausea and vomiting rare

5. Fever—little if any
6. Leucocytes normal or only slightly increased
7. Sedimentation rate is normal
8. Friedman test negative

Oophorrhagia, a term recently introduced by Costallo to designate bleeding from the ruptured Graafian follicle or the corpus luteum, is receiving a deservedly increasing amount of attention. The occurrence of low pelvic pain, not often severe, midway between menstrual periods with little other symptomatology, strongly suggests bleeding from a ruptured follicle. A similar clinical picture developing a week or ten days before the expected date of menstruation indicates the probability of rupture of the corpus luteum.

Although the bleeding into the peritoneal cavity may occasionally be profuse, rarely is it sufficient to give rise to constitutional signs of hemorrhage. There is, as a rule, little if any rise of temperature; the leucocyte count if at all elevated, is only slightly so.

The absence of typical signs and symptoms of the more frequent lesions, together with the characteristic relationship to the menstrual cycle constitute important diagnostic aids. Differentiation of oophorrhagia from ruptured ectopic pregnancy is indicated by absence of the history and the objective findings of pregnancy, by a less critical clinical picture, and by a negative prolan test for pregnancy.

ECTOPIC PREGNANCY

Chief Diagnostic Points

1. Occurs usually after missed period
2. Onset is sudden
3. Pain is severe
4. Shock often supervenes
5. Constitutional signs of hemorrhage
6. Signs on examination suggest pregnancy
7. Extreme tenderness of cervix
8. Cul-de-sac tenderness
9. May or may not feel mass
10. Leucocyte count varies with attacks of pain
11. Sedimentation rate only moderately rapid
12. Positive Friedman test

Schumann, in his monograph on ectopic pregnancy, estimates that somewhat more than three-fourths of patients with ectopic pregnancy give the history of some irregular menstrual bleeding. In most of these the bleeding follows a period of amenorrhea which may vary from a few days' delay of menstruation to several weeks of absence. The bleeding is most often not profuse, is dark in color and is almost invariably associated with attacks of pelvic pain. Profuse external bleeding in a case of suspected ectopic pregnancy tends to indicate that the pain is more likely due to a threatened or incomplete abortion of an intra-uterine pregnancy. The close association of the bleeding with attacks of pelvic pain is, of course, attributable to the fact that the uterine decidua becomes detached as a result of death of the embryo incident to its separation from the tubal wall.

The pain in ectopic pregnancy is characteristically more sudden in onset and of greater severity than that encountered in any other strictly pelvic condition; so much so, that this point alone should arouse the suspicion of tubal pregnancy. Painful defecation and dyspareunia are quite commonly found in older cases in which a pelvic hematocele has formed.

The signs of shock and hemorrhage, particularly when superimposed on the clinical picture just described, leave little doubt of the correct conclusion. The leucocyte count characteristically varies directly with the occurrence of hemorrhage into the peritoneal cavity, being often quite high shortly after the onset of an attack, only to subside almost, if not quite, to normal between episodes of bleeding. The sedimentation rate, in our experience, has been relatively slow unless determined during or shortly after massive internal bleeding.

On examination one usually finds signs suggesting early pregnancy: a succulent vagina, a soft and bluish cervix, together with enlargement of the uterus often to the

size of a two months' normal pregnancy. Polak always stressed the extreme, even exquisite, tenderness of the cervix, particularly on motion toward the symphysis, as almost pathognomonic of ectopic pregnancy. Excruciating tenderness in one or the other lateral areas is the only consistent adnexal finding. Soon after rupture or tubal abortion has occurred, the consistency of the mass and the free intraperitoneal blood is such that extreme tenderness is, indeed, the only positive finding. In the older cases, however, in which some degree of organization has occurred, masses of considerable size can be easily detected in the cul-de-sac and adnexal areas.

The Friedman test, if positive, in a typical case, is undoubtedly of great aid in establishing the diagnosis. Dependent as it is, however, on the presence of functioning chorionic tissue, the test is unfortunately negative in many cases of ectopic pregnancy. In doubtful cases exploration of the cul-de-sac through the posterior vaginal vault, either by aspirating needle or incision, is of invaluable aid, particularly in differentiating between ectopic pregnancy and inflammatory disease.

TWISTED OVARIAN CYST

Chief Diagnostic Points

1. Previous knowledge or suspicion of cyst
2. No history or signs of infection
3. No history suggesting pregnancy
4. Shock often profound
5. Fever and leucocytosis are delayed
6. Sedimentation rate little affected
7. Red cell count not affected unless hemorrhage supervenes
8. Discovery of cyst on examination

In those cases in which a cyst has previously been known to exist the diagnosis of a twist of the pedicle is relatively simple. The same is true also in those cases in which, on examination, a large cyst can be distinctly palpated. On the contrary, cysts of smaller size, particularly when tender as result of a twist, are easily mistaken for ectopic pregnancy; this is especially true when the twisted cyst complicates an early intra-uterine pregnancy. The pain of the twisted cyst is usually not so intense as in ectopic pregnancy; vaginal bleeding is rarely associated with it and the degree of shock is usually not so profound. The absence of a rapid drop in the red cell count in a shocked patient would tend to suggest a twisted cyst as opposed to ectopic pregnancy. On the other hand, the rapid development of anemia might well be due to hemorrhage into the twisted cyst. Since, in such cases, differentiation is well nigh impossible, it is indeed fortunate that immediate laparotomy is imperative in either event.

URETERAL STONE

Chief Diagnostic Points

1. Severe colic
2. Urinary reference of pain
3. Frequency and urgency
4. Red cells in urine
5. Positive *x*-ray and cystoscopic findings

Pain of intense lancinating severity should at once suggest the possibility of a stone in the ureter. The gynecologist who is aware of the ease of mistaking lesions of the urinary tract for those more strictly of pelvic origin will make more frequent use of consultation with the urologist in suspicious cases. Since the methods of diagnosis of stone are almost positively conclusive, it seems unnecessary in this presentation to discuss them at any length.

In closing this brief discussion the author reiterates the necessity of evaluating the clinical picture as a whole rather than depending on any one sign or symptom as pathognomonic.

DIAGNOSIS OF DEHYDRATION IN SURGICAL CONDITIONS*

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THERE are four main types of fluid in which the body is likely to become deficient. These are: (1) Water, (2) extracellular fluid (water + salt), (3) plasma (water + salt + protein), and (4) blood (water + salt + protein + cells).

Loss of plasma and loss of blood are not commonly referred to as "dehydration" and will not be included in the present discussion. Blood shortage is generally spoken of as "hemorrhage" or "anemia." Plasma shortage has no particular designation, except when it results in shock, in which case it may be referred to as "burn shock" or "wound shock." Occasionally, plasma loss is referred to as "dehydration." However, in the present discussion we shall deal only with conditions arising from shortage of water and shortage of salt. We shall deal, therefore, with two conditions, both of which are commonly referred to as "dehydration," but which, as will be pointed out, are quite dissimilar.

First we shall review a few fundamental facts concerning the distribution and metabolism of water and salt in the body. Then we shall consider the manifestations which arise when the body is deficient in these substances.

DISTRIBUTION OF WATER AND SALT IN THE BODY

About 70 per cent of the body is water.[1] Since all body membranes are freely permeable to water, this substance distributes itself in accordance with the laws of osmotic equilibrium. For the purposes of this discussion there are three important fluid compartments in the body, each of which contains fluid of a characteristic type: Intracellular water accounts for about 50 per cent of the total body weight (or 70 per cent of the body water). Intracellular fluid is relatively rich in potassium, but contains little or no sodium or chloride.[1] Extracellular fluid, which is relatively poor in potassium and rich in sodium and chloride, consists of two parts, namely, the blood plasma and the interstitial fluid. The blood plasma contains about 7 per cent of protein, while the interstitial fluid is practically protein free.

Thus, the distribution of water between intracellular and extracellular compartments depends, other things being equal, upon the amount of sodium in the body.[1] (The amount of chloride in the body is of only minor importance, as a chloride deficiency is readily compensated for osmotically by an increase in bicarbonate. Sodium, on the other hand, which constitutes about 90 per cent of the extracellular base, is not replaceable to any significant extent except by sodium itself.) Similarly, the distribution of extracellular fluid between plasma and interstitial compartments depends, other things being equal, upon the amount of protein in the plasma. The inorganic extracellular electrolytes, such as sodium, pass freely through the capillary walls and, therefore, can have little if any effect upon the distribution of water between the plasma and the interstitial compartments.

DAILY REQUIREMENTS OF WATER AND SALT

The normal person can tolerate total abstinence from salt for days and perhaps weeks without ill effects. The excretion of sodium and chloride in the urine under such circumstances practically ceases and the losses from the body become negligible. A salt deficiency is not likely to develop unless there is a definite abnormal loss,

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such as occurs in vomiting, diarrhea, profuse sweating or Addison's disease.

Total abstinence from water, on the other hand, cannot be tolerated for more than a few days. The body possesses no mechanism for preventing the inexorable daily losses of water. A liter or more of water is frequently lost daily by evaporation from the skin and lungs, even in a dehydrated patient.

The contrast between salt and water requirements may be stated in a different way: Loss of water, both sensible and insensible, is taking place all the time so that there is a *daily* need for relatively large quantities of water even when there is no abnormal water loss. Need for salt, on the other hand, arises as a rule, only when there has been some definite *abnormal* loss. And once this salt loss has been made good, assuming that the abnormal loss has ceased, administration of additional salt is useless and may in some cases be actually harmful. Normally, the kidneys will excrete promptly an excess of either water or salt.

In the case of salt we may properly think of the daily intake as an independent variable and the output as a dependent variable. In the case of water the daily outgo is, up to a certain point, the independent variable while the intake is the dependent variable, insofar as the water intake is governed by the sensation of thirst. The mechanism of thirst practically guarantees a normal water intake in a normal individual. There is no similar mechanism to guarantee a normal salt intake, nor need there be under ordinary circumstances, for it is a most unusual diet that does not contain an excess of salt.

TWO TYPES OF DEHYDRATION

Dehydration is of two distinct types or some combination of the two, depending upon whether it is due to (1) inadequate water intake, (2) abnormal fluid loss, or (3) both of these factors working simultaneously.[2] At first glance it would appear entirely immaterial whether dehydration results from one or the other of these factors; the net result might be presumed to be the same. Such, however, is not the case.

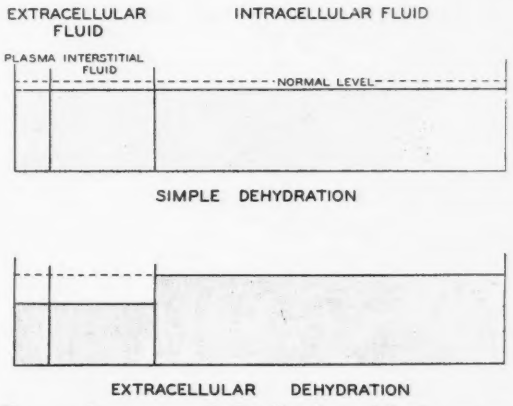

Fig. 1. Amount and distribution of body water in simple dehydration and in extracellular dehydration.

When dehydration results from inadequate water intake the shortage of water is not accompanied by an abnormal loss or salt. Therefore, the deficiency of water is felt in all parts of the body, within the cells as well as in the extracellular compartments. There occurs no maldistribution of water in this condition because there is no significant change in the amount or distribution of dissolved substances. The manifestations consist of *thirst*, oliguria, concentrated urine, and a rising blood nonprotein nitrogen.

When dehydration results, on the other hand, from abnormal fluid loss, there occurs a simultaneous loss both of water and of extracellular electrolytes, chiefly sodium and chloride. The loss of sodium results in a corresponding loss of extracellular water, and there occurs a diminution in extracellular fluid volume (including plasma volume) even though the water intake is normal. As the condition advances, manifestations of circulatory failure develop. There occur weakness, apathy, anorexia, fainting, hypotension, shock, anuria and death. Before any of these symptoms are apparent, there occurs a progressive hemoconcentration. *Thirst is not present.* The urinary output may be normal until the stage of circulatory failure

is reached. Since this condition is characterized by a reduction in extracellular volume (including, of course, the plasma volume), we have referred to it as "extracellular dehydration" or "extracellular desiccation," in contrast to "simple dehydration." In Figure 1, the essential difference between the two types of dehydration is diagrammatically illustrated. In Table 1 are listed the outstanding manifestations of these two distinct entities.

DIAGNOSIS OF SIMPLE DEHYDRATION

When seen in pure form, simple dehydration presents no particular problem in diagnosis. The condition arises whenever an individual for any reason cannot obtain, cannot ingest or cannot absorb the water which he needs. The resulting manifestations are thirst, dry mouth, oliguria, concentrated urine and a rising blood nonprotein nitrogen. (Table 1.)

TABLE 1
MANIFESTATIONS OF THE TWO TYPES OF DEHYDRATION WHEN SEEN IN PURE FORM

Simple (True) Dehydration	Extracellular Desiccation
Thirst	Weakness and apathy
Dry mouth	Anorexia or nausea
Oliguria	Low blood pressure or shock
Concentrated urine	Hemoconcentration
Rising nonprotein nitrogen	Often hypochloremia
	Sometimes anuria and rising nonprotein nitrogen if blood pressure sufficiently low

The symptom of thirst is a very sensitive indication of the need of the body as a whole for water. There is evidence that thirst is closely related to *cellular* dehydration.[3] Oliguria, but not anuria, is also a cardinal manifestation of simple dehydration (except in diabetes insipidus). The azotemia of dehydration is due chiefly to the associated oliguria.

The degree of dehydration may be difficult to judge in some instances. In general, the degree of dehydration seems to be in direct proportion to the intensity of thirst, and it is unfortunate that thirst does not lend itself to quantitative measurement. The degree of oliguria decidedly is not an accurate index of the extent of dehydration.[2] A moderately, and also a severely, dehydrated person will generally excrete around 400 cc. of urine per day. The level of blood nonprotein nitrogen may be of some aid, provided too much emphasis is not placed upon this one finding. Other things being equal, the higher the level of the blood nonprotein nitrogen, the longer has been the duration and the greater is the extent of the dehydration.

Coller and Maddock found that subjects placed on a dry diet developed nitrogen retention and severe thirst in three or four days, by which time they had become dehydrated to the extent of 6 per cent of their body weight.[4]

At the present time there exists no perfect clinical method for estimating the degree of dehydration. Although the McClure-Aldrich test has been recommended by some, a recent study carried out by MacIntyre, Pedersen and Maddock found the test to be unreliable.[5]

From the practical standpoint, it is not necessary to make a quantitatively accurate diagnosis in cases of simple dehydration. In any case, one must administer water (or glucose solution) until the manifestations of dehydration have completely disappeared, that is, until thirst is relieved and diuresis is established (at least 1,000 cc. of urine daily), and until the blood nonprotein nitrogen has returned to normal.

DIAGNOSIS OF EXTRACELLULAR DEHYDRATION

The diagnosis of extracellular dehydration is not always a simple matter. It is easily overlooked, especially in the earlier stages when there are no striking signs or symptoms to announce its presence. The symptoms of weakness, apathy and anorexia which precede the onset of circulatory collapse, are likely to be attributed erroneously to other causes.

Extracellular dehydration must be thought of and watched for in any patient who is losing electrolytes abnormally,

whether from the skin (profuse sweating), from the gastrointestinal tract (vomiting, diarrhea, fistula), or through the kidneys (Addison's disease). Among surgical patients extracellular dehydration is most likely to arise among those who are losing fluid abnormally from the gastrointestinal tract. The sequence of events is likely to be somewhat as follows: A patient with profuse drainage from a biliary fistula or from an ileostomy is enjoying an apparently normal postoperative convalescence, is drinking plenty of water and is excreting a good volume of urine. About seven to fourteen days after the operation, he complains of weakness and loss of appetite and perhaps nausea and vomiting. A day or two later it is discovered that the patient has not voided for twenty-four hours. Catheterization yields only 20 cc. of urine. The pulse is found to be weak and the blood pressure is discovered to be 60 mm. of mercury systolic. The plasma chloride concentration is 460 per cent (normal 560) and the blood nonprotein nitrogen is 60 mg. per cent. The intravenous administration of three or four liters of saline or Ringer's solution results in dramatic recovery, if carried out promptly. The daily administration, orally or parenterally, of an amount of sodium chloride equivalent to the daily losses (usually 5 to 20 Gm.) will prevent recurrence. In one such case personally observed by the author it was necessary to administer over 20 Gm. of sodium chloride daily in order to replace the losses (ileostomy) and to prevent the recurrence of shock.

When extracellular dehydration progresses to the point of shock, we have occasionally referred to the condition as "electrolyte shock." In the past the condition frequently has been referred to as "hypochloremic shock," but this is a misleading term for "hypochloremia" bears no essential relationship to the condition in question. If, for instance, extracellular dehydration results from diarrhea, the plasma chloride concentration may be normal, or even increased, and yet the patient may be in dire need of sodium chloride solution. Of course, the prime need is for sodium, the chloride playing an incidental rôle. The shock which occurs is indistinguishable clinically from other types of hematogenic shock. It results from a diminished blood volume, as the associated hemoconcentration indicates.

Table II

Conditions likely to give rise to the different types of dehydration

True dehydration (Loss of water)	Simple water deprivation Marked dysphagia Marked anorexia
Extracellular desiccation (Loss of sodium chloride and water)	Diarrhea (e.g., cholera) External fistulas Addison's disease Profuse sweating
Mixed type	Intestinal obstruction Vomiting from any cause Wangensteen suction

To make the diagnosis of extracellular dehydration before circulatory collapse occurs, one must depend upon clinical and laboratory observations, including determinations of the blood pressure, hematocrit, plasma protein concentration, plasma chloride concentration and carbon dioxide combining power. If the blood pressure is low, circulatory collapse should be considered imminent; but before the blood pressure falls there will be found a progressive hemoconcentration. Also there will generally be found some alteration in the plasma chloride concentration and often in the plasma carbon dioxide combining power, depending upon the composition of the fluid which has been lost from the body.

One must, of course, be cautious in the evaluation of hemoconcentration. One patient observed by the author had had a recent ileostomy performed for chronic ulcerative colitis and was losing between four and five liters of liquid feces daily. This patient went into shock and was found to have a hematocrit of 53.3 per cent and a plasma protein concentration of 8.0 per cent. (Table III.) The administration of fluid containing 31 Gm. of sodium chloride resulted in dramatic improvement and a reduction in the hematocrit and plasma protein concentration to 41.4 per cent and

6.5 per cent, respectively. During the following day, however, only 8 Gm. of sodium chloride were administered and the patient developed mild shock. The hematocrit and plasma protein concentration were found to have risen again. (Table III.) The administration of fluid containing 38.5 Gm. of sodium chloride completely relieved the symptoms once more and resulted in a return of the hematocrit and plasma protein concentration to what were probably their true levels (35 per cent and 5.4 per cent, respectively). It is thus important to bear in mind that if a patient has anemia and hypoproteinemia before hemoconcentration occurs, the hematocrit and plasma protein concentration may not rise above the normal level, though the patient may be in desperate need of saline therapy. In such cases the diagnosis is more difficult and it is probably wise to play safe and supplement the saline therapy with transfusions of blood or plasma and it is certainly wise to watch closely for the development of edema.

hydration. When the sodium losses are great, the corresponding losses of extracellular water with resulting hemoconcentration may mask the shortage of chloride. Our chemical determinations do not tell us how much chloride has been lost from the body but merely inform us of the chloride *concentration* in the specimen examined. Also, in evaluating the results of saline therapy one must not expect the plasma chloride concentration to return promptly to normal except in those cases in which the disease is of short duration. Several days may be required to accomplish this. (Table III.)

DIAGNOSIS OF MIXED TYPE OF DEHYDRATION

It is very common for the two types of dehydration to co-exist in the same patient. Vomiting commonly gives rise to the mixed type because a patient who is vomiting not only is losing sodium and chloride but also is generally unable to drink a normal amount of water. In such cases each of the two types of dehydration must be diagnosed on its own merits. This should cause no difficulty because the two conditions have practically nothing in common.

TABLE III*

Days	Intake		Output (Urine), Cc.	Blood			Remarks
	Water, Cc.	Sodium Chloride, Gm.		Hematocrit, Per Cent	Plasma Protein, Gm. Per Cent	Plasma Chloride Mg. Per Cent (as Sodium Chloride)	
0	....	....	....	53.3	8.0	460	In shock
1	4925	31.0	1050	41.4	6.5	507	Improved
2	2825	8.0	400+	48.7	6.8	465	Mild shock
3	6000	38.5	1030+	35.1	5.4	508	Marked improvement
4	4900	26.0	670	34.9	5.3	512	Feels well; plasma transfusion 500 cc.
5	3800	23.4	960	34.3	5.3	498	Feels well; plasma transfusion 400 cc.
6	3600	19.5	1420	36.3	5.6	508	Feels well
7	5495	23.8	980	38.8	5.5	...	Feels well; blood transfusion 500 cc.
8	4250	21.0	780+	33.6	4.7	578	Feels well

* Patient L. S. Ileostomy for chronic ulcerative colitis. Extracellular dehydration and shock from loss of large amounts of fluid containing extracellular electrolytes.

Determinations of plasma chloride concentration are of definite aid in diagnosis, but it should be remembered that normal values do not rule out extracellular de-

A patient who has been vomiting or who has both diarrhea and vomiting or a patient with intestinal obstruction should automatically be suspected of having the mixed type of dehydration. The simultaneous presence of thirst, oliguria, weakness, apathy and anorexia make such a diagnosis highly probable. The diagnosis will be made more certain by the finding of hypotension or shock, an elevated hematocrit and plasma protein concentration, a rising blood nonprotein nitrogen, and a lowered plasma chloride concentration.

If mixed dehydration is not recognized and is treated either by physiological saline alone or by glucose solution alone, the dehydration may then become converted into one or the other of the pure types. The author has seen this actually happen.

The common occurrence of the mixed type of dehydration probably explains the failure of most observers in the past to distinguish clearly between the two component types.

SUMMARY

1. Some fundamental facts concerning the distribution and turnover of water and salt in the body are reviewed.

2. Simple dehydration and extracellular dehydration are discussed with respect to mechanism of production, symptomatology and diagnosis.

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VITAMIN THERAPY IN THE SURGICAL PATIENT*

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RECENT experimental and clinical literature has continued to emphasize the importance of adequate vitamin maintenance in pathological conditions of an obviously surgical nature. This emphasis has largely risen from repeated demonstrations that deficiency in certain if not all of the accessory food factors produces local and constitutional changes of such nature that surgical treatment attempted in the presence of deficiency may become more hazardous and recovery imperilled. The purpose of this communication is to reiterate several aspects of vitamin therapy in general and correlate their importance to surgical treatment in particular.

To facilitate the discussion, all vitamin complexes may be divided arbitrarily into two groups: (1) Vitamin factors essential in adequate amounts for maintenance of normal health and nutrition but deficiency of which is not of primary surgical importance; (2) vitamin factors essential in adequate amounts for maintenance of normal health and nutrition but deficiency of which directly increases surgical risk. However vitamin factors are arbitrarily divided or classified, certain well established clinical concepts are common to all as a general nutritional group. A general consideration of these concepts is essential to a more specific discussion of those having particular surgical consequence.

Definite amounts of all vitamins are required for complete maintenance of body nutrition and function. With an adequate diet, under normal conditions of digestion, more than is requisite is available. When, however, the diet becomes inadequate in vitamin yielding substances, or the physiological processes making them available become impaired by disease, a deficiency in the body vitamin content is the result. Such deficiency may exist in one or all of the vitamin substances and, when of sufficient severity and duration, is reflected by impairment of various body tissues and functions. Ultimately, if unrelieved, such deficiency becomes apparent by ordinary clinical methods. But subclinical deficiencies are of far more frequent occurrence. It is not to be presumed, therefore, that because the degree of vitamin deficiency is not outwardly apparent, no deficiency exists. For it has been substantially proved that deficiency of such a degree to cause marked alteration in tissue functions and structure can be extant without grossly detectable change. It should not be inferred from the foregoing that every individual must have one or more vitamin deficiency. It is implied, however, that every individual case may have a potential deficiency. Deficiency states do not develop in normal individuals taking normal well balanced diets. They are, however, particularly prone to develop in the following commonly encountered groups in surgical practice:

1. Individuals on restricted dietary régimes indicated in the treatment of incidental disease, e.g., diabetes, peptic ulcer, obesity.

2. Individuals taking pharmacological agents into the gastrointestinal tract which inhibit absorption or destroy the vitamin substances ingested. Examples of these instances are frequent. It is generally conceded that excessive ingestion of mineral oil prevents absorption of the fat soluble vitamins A, D and K.[1,2] Alcohol in excess

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notably results in decreased ingestion of all food and of B complex in particular.[3,4,5]

3. Individuals in which a particular disease, usually of the gastrointestinal tract or its attendant secretory organs, precludes the absorption of the vitamin substances ingested or after absorption interferes with proper utilization. An example of this condition is vitamin K deficiency in gallbladder disfunction causing lack of bile for the emulsification and absorption of fatty substances with consequent reduction in the vitamin K level of blood and tissues.[6,7,8]

It should be borne in mind constantly that vitamin substances are, individually and as a group, specific nutritional identities, each of which produces changes more or less specific in body tissue and functions, and that the pathological changes induced by the deficiency respond specifically to adequate amounts of the proper vitamin substance. This specificity together with pertinent therapeutic addenda is recalled in Table 1:

two groups: The first of these includes those vitamins of minor or indirect surgical importance, the second includes those of major surgical interest.

Group I. Vitamin Factors of Minor Importance in Surgical Treatment. The constituents of this group include vitamins A, D and E which for convenience and clarity are considered individually.

VITAMIN A

Vitamin A is a fat soluble, water insoluble, crystalline substance closely allied though not identical with the compound β carotene. It is of wide distribution in plant and animal bodies and occurs in large amounts in pigment and vegetable substances. The vitamin itself is a primary alcohol of which β carotene is the predominant precursor, which is converted in the liver to the vitamin proper. Two important physiological functions depend upon the presence of sufficient quantities of the vitamin.[8,9,10]

1. Visual adaptation to darkness which

TABLE I*

VITAMIN	CHEMICAL SUBSTANCE	PHYSIOLOGICAL FUNCTION	DEFICIENCY SYNDROME	APPROXIMATE ADULT DAILY REQUIREMENT
A	BETA-CAROTENE	VISUAL ADAPTATION TO DARK. INTEGRITY OF BODY EPITHELIUM.	NIGHT BLINDNESS. KERATITIS & EPITHELIAL ULCERS	8000 UNITS
B	THIAMIN	ESSENTIAL ELEMENTS IN CELLULAR OXIDATION	NERVOUS & CARDIO-VASCU- DISORDERS (BERI-BERI).	30 UNITS
B	RIBOFLAVIN	SAME.	LESIONS OF BUCCAL MUCOSA.	3 MGMS.
B	NICOTINIC ACID	SAME	CEREBRAL, GASTRO-INTESTINAL & SKIN DISORDERS (PELLAGRA)	150 MGMS
B	OTHER FACTORS	———	———	———
C	ASCORBIC (CEVITAMIC) ACID	COLLAGEN FORMATION & CONNECTIVE TISSUE CELL PROLIFERATION	ABNORMALITIES IN CONNECTIVE TISSUE STRUCTURE OF BODY TISSUES & HEALING PROCESS (SCURVY).	120 UNITS
D	CALCIFEROL (VIT. D2) ACTIVATED DEHYDRO-CHOLESTEROL (VIT. D3)	Ca ABSORPTION FROM GASTRO-INTESTINAL TRACT & RENAL Ca & P EXCRETION.	BONY ABNORMALITIES. RICKETS. DELAYED BONE HEALING	200 UNITS
E	ALPHA-TOCOPHEROL	GESTATION & STRIATE MUSCLE IN EXPERIMENTAL ANIMALS	NOT DEFINITE IN HUMANS.	NOT DETERMINED
K	2-METHYL-3-PHYTYL-1, 4-NAPHTHOQUINONE	PROTHROMBIN FORMATION IN BLOOD CLOTTING PROCESS.	DELAYED CLOTTING TIME HEMORRHAGE.	1 MGM

*Compiled by authors from various sources.

It was previously stated that to facilitate discussion of these vitamin factors of particular consequence from the surgical standpoint, a division of the entire group of vitamins would arbitrarily be made into

is dependent on speed of breakdown and resynthesis of the visual purple (rhodopsin) substance of the retina. Rhodopsin is apparently a compound composed of a protein and a prosthetic group presumed to

be vitamin A. In deficiency states the ability of dark accommodation is rapidly and severely affected giving rise to the condition of night blindness or nyctalopia. This condition can usually be rapidly corrected by the administration of adequate vitamin substance.

2. The integrity of epithelial structure and function is maintained only in the presence of normal body vitamin A content. The physiological processes which condition the presence of the vitamin has not as yet been elucidated. But lack of the vitamin has been shown to produce epithelial atrophy with subsequent proliferation of the basal cells and replacement with a modified epithelium. This deficiency effect is particularly evident in the epithelium of the cornea, skin, respiratory tract, urinary passages and mucosa of the gastrointestinal tract. Of consequence corneal and skin lesions, increased susceptibility to respiratory tract inflammation, urinary calculi and gastrointestinal pathological conditions arise from the more advanced deficiency states.

The average daily minimum requirement is in the neighborhood of 2,000 international units. The unit standard is reckoned on a basis of the quantity of the vitamin required to promote normal growth in rats of a standard age and weight fed on an A deficient diet over a specific time interval. The international unit of which the U.S.P. unit is the equivalent represents the physiological activity of .006 mg. of purified β carotene. During periods of growth and pregnancy or in deficiency conditions the dosage of vitamin A should be proportionately increased.

From the foregoing consideration of vitamin A substance it can be concluded that the relation of this vitamin and the deficiency states arising from its lack have only a rather remote and indirect application to surgical therapy. Deficiency in vitamin A may serve as an index to a coincident deficiency in the other fat soluble vitamins, D, K and E, since they are also fat soluble and are influenced by the same common conditions of dietary content and absorption. It is moreover conceivable that in severe degrees of vitamin A depletion the epithelial changes produced may interfere considerably with tissue repair after surgery, either because of atrophic changes which occur or susceptibility to secondary infection.

VITAMIN D

Numerous sterol substances of plant and animal origin when irradiated with ultra violet light are changed in internal structure so that they possess vitamin D activity.[11,12] Of these substances the most important are: (1) Calciferol or vitamin D_2 produced by the irradiation of ergosterol; (2) dihydrotachysterol formed by the reduction of tachysterol which is another of the products of the progressive radiation of ergosterol; and (3) activated dihydrocholesterol or vitamin D_3 synthesized by the irradiation of 7-dihydrocholesterol.

Two of the three foregoing substances, namely, calciferol and activated dihydrocholesterol, though not identical in chemical structure and physiological activity, are sufficiently similar to be classed under the general term of vitamin D. The third substance, dihydrotachysterol, differs considerably in its physiological action and will be separately considered.

Vitamin D is one of the fat soluble vitamin group. It can be made available to the body in adequate quantities only when it is present in sufficient amounts in the diet and under conditions of adequate fat absorption from the digestive tract. When the preparation for or absorption of fatty substances of the diet are impaired from whatever cause, D deficiency is apt to result. The vitamin D factor is closely related to the body phosphorus and calcium. Together with the parathyroid glands it regulates to a large measure the calcium and phosphorus blood levels. The principal action of vitamin D in this relationship is to facilitate the absorption of calcium from the gastrointestinal tract. Beside being essential for calcium absorp-

tion, it apparently is capable of producing excretion of phosphorus by the kidney, which, if continued, results in withdrawal of calcium from the bones. In this latter action, vitamin D resembles the active secretion of the parathyroid which mobilizes calcium from storage in the bones but does not influence its absorption from the digestive tract. The typical rachitic lesions of vitamin D deficiency are produced by the inability of the body to absorb sufficient calcium from the gastrointestinal tract. In this state the blood calcium level would markedly fall if calcium were not withdrawn from the bone calcium deposits to maintain it. The withdrawal of calcium from the bone to maintain the calcium blood level results in rickets or osteomalacia with consequent deformity. In contrast it is possible to produce the reverse of deficiency conditions by the administration of enormous amounts of active vitamin D substance. Under these circumstances there is considerable elevation of the blood calcium and phosphorus level at the expense of bone deposits, with the formation of calcareous concretions in the gastrointestinal tract, vascular system, renal parenchyma and liver.

The action of dihydrotachysterol, although a related member of the vitamin D group, differs quantitatively in action from the two previously mentioned substances. It facilitates calcium and phosphorus absorption from the gastrointestinal tract but its greatest action is upon calcium excretion. Dihydrotachysterol produces a marked increase in phosphate excretion by the kidney which causes a concomitant renal excretion of calcium. To maintain the calcium blood level, skeletal calcium is liberated into the blood. This mobilization of bone calcium is in many respects similar to parathormone, the secretion of the parathyroid gland which also mobilizes calcium and maintains the blood level by a similar mechanism.[13]

Vitamin D deficiency states are reflected in low blood calcium levels and in abnormalities of bones, resulting in the typical rachitic syndrome. Such deficiency is particularly prone to occur in infancy and early childhood, critical periods of bony growth, in pregnancy when the vitamin requirements are considerably increased, and in the aged in whom the dietary intake is apt to be abnormally low.[14,15]

Considerable variation in the daily requirements exist therefore depending on the age of the patient. Infants on artificial diets should have 500 to 1,000 units daily. Breast fed infants on whole cows milk régime 250 to 500 units and older children ought to receive a similar amount. In adults, particularly those of sedentary occupation, supplemental vitamin D may be of benefit in amounts of 150 to 200 units. During pregnancy and lactation, quantities are essential in the neighbourhood of 1,000 units. In aged individuals, suffering from bone injuries, the administration of 100 to 200 units daily depending on the diet, may be considered advisable. When severe deficiency exists, the dosage must be greatly increased, and in these instances daily amounts of from 20,000 to 30,000 units may be employed. In all instances in which vitamin D is given, an adequate calcium intake should be assured.

The international unit on which vitamin D administration is based has been agreed upon as the physiological activity of .0025 mg. of crystalline calciferol.

Indications for the use of vitamin D may be included in the following: (1) All cases of suspected deficiency. In these instances the dosage is required in therapeutic amounts; (2) prophylactically in infants, children, pregnant or lactating women and in individuals of sedentary indoor habits. (3) cases of bone injury, particularly in the aged; (4) Gastrointestinal diseases in which fat absorption is apt to be inadequate and, therefore, reduce the availability of vitamin D.

VITAMIN E

In the present state of investigation, only a passing notice need be taken of vitamin E. This arises from the fact that its indis-

pensability in the human has not been proved and information regarding it rests almost entirely upon a basis of animal experimentation.

Vitamin E is a fat soluble substance existing in several tocopherol forms. Of these forms, alpha tocopherol possesses the greatest activity and is readily inactivated by oxidation.[16,17]

Symptoms of deficiency are notably shown by the effect on gestation and the effect on striate muscle in rats. Clinical application of vitamin E in the human is, as previously suggested, extremely limited. It has been advanced as a form of therapy in habitual abortion, certain degenerative diseases of the nervous system and in muscular dystrophies. Its efficacy in these conditions is doubtful and lacks confirmation.

VITAMIN P

Vitamin P (lemon eroidictin) is a recently isolated substance. There are disputed claims that it is of value in the treatment of purpura. To date the value of its use in man has not been established.[18]

Group II. Vitamin Substances of Major Surgical Importance.

VITAMIN K

The discovery and clinical application of the vitamin K factor represents one of the major recent advancements in medical research.[19,20] Its importance in surgical treatment can hardly be overemphasized, since it has greatly lowered the operative risk and mortality in one large group of diseases for which surgery in some form is the accepted essential treatment. The excellent results obtained in vitamin K therapy are based upon an increasingly intimate knowledge of its chemistry and physiological activity and a recognition of the pathological conditions in which its use is specifically indicated.

Investigations of its chemical nature have demonstrated that it is a fat soluble compound containing a quinone structure that is present in a wide variety of vegetable and animal substances, notably in certain plants and in animal liver.[21] Further experimentation has shown that a large number of substances possess various degrees of vitamin K activity, but that vitamin K proper was not a single chemical entity but occurs in the natural state in the form of at least two separate compounds of related chemical structure. The substance selected as representative is 2 methyl-3 phytyl-1, 4 naphtho quinone or vitamin K.

Under normal physiological circumstances, articles of diet containing vitamin K are taken into the gastrointestinal tract where they are subjected to the action of the various digestive processes and the fat elements containing the K fraction liberated. This K containing fat fraction is mixed with and emulsified by bile and absorbed by the intestinal mucosa into the blood by which it is carried to the liver for immediate use and storage. The function of the vitamin K fraction is to provide an element in the formation by the liver of an essential blood clotting factor, namely, prothrombin. Prothrombin is then liberated into the blood in adequate quantities for normal clotting. The relationship of prothrombin to the other elements in blood coagulation process will be recalled by the following brief schema:

$$\text{Thromboplastin} + \text{prothrombin} + \text{calcium} \rightarrow \text{thrombin}$$

$$\text{Thrombin} + \text{Fibrinogen} \rightarrow \text{fibrin (clot)}.$$

Lack of any one of these essential elements inhibits the clotting process in direct proportion to the deficiency present. When all are present in sufficient quantity coagulation occurs normally. Deficiency in prothrombin is at once reflected by a clotting time prolonged in proportion to the extent of the deficiency present. Since the clotting process depends upon the presence of adequate quantities of prothrombin, and since prothrombin can be formed in adequate amounts only when there is absorption and utilization by the liver of requisite

amounts of vitamin K factor, it is obvious that the normal clotting process is ultimately dependent upon adequate amounts of vitamin K. From the foregoing it will be readily perceived that vitamin K and prothrombin deficiency can occur in any one of the following conditions: (1) Insufficient vitamin K in the diet; (2) Impaired absorption resulting from: (a) insufficient bile for conversion into an absorbable state; (b) abnormalities of the absorptive intestinal surface resulting from disease; (3) impaired liver function, resulting in the inability to form prothrombin.

Hence it would be expected that in inadequate diets, diseases of the biliary system, particularly those in which the flow of bile to the intestine is reduced or abolished, diseases of the gastrointestinal tract which interfere with the absorption of digested nutritional elements, and pathological conditions of the liver of such a nature that its function is impaired, would result in abnormal delay of the blood clotting process. This is actually the case.

Considered on the physiological basis outlined above, there are several groups of conditions which are intimately related to vitamin K and prothrombin deficiency:

1. The first group of patients with prothrombin deficiency to be considered are newborn infants.[22,29] This deficiency of prothrombin is apparently physiological and occurs in all infants, it being about 60 to 70 per cent of the normal adult value. There may be a marked fall twenty-four hours after birth which is usually of short duration. A spontaneous rise usually begins one or two days later, possibly due to the fact the infant begins to take food and bacterial flora are established which synthetize vitamin K. If spontaneous recovery is delayed, hemorrhagic disease develops. In such instances the prothrombin level may be as low as 5 per cent of normal, which may be corrected rapidly by the administration of vitamin K.

2. The second group of prothrombin deficiencies are those due to an inadequate intake of vitamin K in the diet. Experimentally, these have not been produced in mammals by means of a vitamin K free diet alone, and in man such a vitamin K deficiency is exceedingly rare. Several cases are reported but other deficiencies were present. The explanation of the rarity of this condition is thought to indicate that bacterial activity in the gut must produce enough vitamin K to meet minimal requirements.

3. The third group of prothrombin deficiencies are those in which for some reason there has been a failure of absorption of K from the gastrointestinal tract. This group is by far the most important one to consider. It is now known that the absorption of a fat soluble vitamin requires the presence of bile of normal composition in the gut. The feeding of fat soluble vitamin K alone in the absence of bile has little or no effect on the control of prothrombin deficiencies, whereas, if bile or bile salts are given with the same amount of vitamin K, there is an immediate increase of prothrombin in the circulating blood.[23,24,25] It is further known that the feeding of bile alone will correct prothrombin deficiencies by facilitating the absorption of vitamin K which is normally present in the gut. In this group then, in which there is a failure of vitamin K absorption, we must first consider:

1. Those cases due to simple exclusion of bile from the gut as produced by a biliary fistula.[26]
2. Those due to obstruction resulting from stones, strictures, tumors, or inflammatory processes.
   (*a*) If complete obstruction exists, vitamin K is not absorbed.
   (*b*) If there is a partial obstruction there may be a decrease in bile salt formation so that the character of the bile may change to such an extent that it will not facilitate the absorption of vitamin K.[23,24]
   (*c*) Hepatic damage; this is a variable factor depending on the nature

and duration of the obstruction and the presence of infection. If severe, the storage and utilization of vitamin K may be interfered with.[24]

3. In certain lesions[27] there is deficiency of prothrombin due to inadequate absorption resulting from unusual losses:
   (*a*) Diarrhea, chronic ulcerative colitis, sprue.
   (*b*) Vomiting in intestinal obstruction, prolonged gastroduodenal suction.
   (*c*) Inadequate absorptive surface, gastrocolic fistula, enterostomies, short-circuiting operations.
4. In the fourth group the prothrombin deficiency is due to primary hepatic damage.[28] Here there is no vitamin K deficiency, but rather there is an inability on the part of the liver to form prothrombin. The ability on the part of the liver to form prothrombin is probably one of the last functions lost. In this group there is a failure to respond to vitamin K (obstructive biliary cirrhosis, advanced acute or chronic parenchymatous disease of the liver).

It has already been pointed out that a number of similar substances possessing the quinone structure show vitamin K activity. No standard universal unit expressing K potency as yet exists but the one in widest use at the present time defines a unit of K in terms of the smallest amount of K substance per Gram weight of experimental chick per day that will produce normal coagulation time in chicks deprived of K after three days of oral administration.

The fat soluble substances containing K factor require bile or bile salts for their absorption from the gastrointestinal tract. Consequently, when these substances are used, bile essential for their utilization must be present either naturally, or therapeutically supplied. Water soluble compounds possessing the K principle may be utilized in the absence of bile and given parenterally.

A review of the conditions of prothrombin deficiencies related to K deficiency is presented below together with specific points on therapy.

TREATMENT

Clinically vitamin K therapy is of value: (1) In the newborn; if vitamin K is administered to the mother intravenously before or during labor, the baby is born with a high level of prothrombin. If surgery is necessary in a newborn infant, vitamin K should certainly be administered.[22,29]

(2) In preparation of the jaundiced patient for surgery vitamin K is of great value. If 2 methyl-1, 4 naphthoquinone (which is fat soluble) is given by mouth together with dried whole bile for three to five days, it will usually bring prothrombin time to normal. Another preparation, the water soluble K_5—(4 amino 2 methyl-1, 4-naphthol hydrochloric acid) may be given by vein.[30] If the response is poor, it is probably the result of extensive liver damage. The treatment should also be continued for several days postoperatively.

(3) In certain intestinal lesions such as intestinal obstruction associated with long continued vomiting, in ulcerative colitis, or polyposis associated with diarrhea; in the presence of a gastrocolic fistula, enterostomy or prolonged gastroduodenal suction, there may be a lengthened prothrombin time resulting from inadequate absorption of vitamin K from the gut. Here parenteral administration of vitamin K is indicated.

Vitamin K has not proved useful in hemophilia, thrombocytopenic purpura, aplastic anemia, acute yellow atrophy or multiple myeloma. In cirrhosis of the liver and in severe hepatitis the response is often poor.

VITAMIN B

It has been demonstrated that vitamin B is not a single chemical and biological identity but is separable in a number of

individual components, each of which differs in chemical structure and physiological action. Although these substances are grouped together under the general term vitamin B complex, for the sake of convenience and as a concession to the older literature, they must be separately considered in any discussion concerning therapeutic values. Not all the individual components comprising the B complex will be enumerated here, but only those of immediate clinical interest. The subsequent discussion will, therefore, be limited to B components, thiamin, riboflavin and nicotinic acid.

THIAMIN

Analysis and synthesis of thiamin have proved it to be an organic compound formed by the union of a pyrimidine and thiazole nucleus.[30] It is a white water soluble crystalline substance readily absorbed from the gastrointestinal tract and rapidly taken up by the tissues. It has been estimated that approximately 10 per cent of the daily intake is metabolized and excreted by the urine. In instances of excessive intake the excretion is correspondingly greater and in the condition of elevated metabolism thiamin is more rapidly destroyed by the tissues and larger amounts are essential for maintenance of normal function.[5] This is particularly evident in hyperthyroidism.

Considerable evidence has accumulated indicating the fact that thiamin is of fundamental importance in the various stages of carbohydrate metabolism. Since practically all tissues of the body utilize carbohydrate in the discharge of their functions, thiamin deficiency is apt to result in marked disturbances throughout the body. Nervous tissue is particularly susceptible to derangement since carbohydrate is practically the sole substance metabolized.

Thiamin deficiency results in a symptom complex (beriberi) chiefly referable to the nervous and cardiovascular systems, with cardiac enlargement and irregularity together with sensory and motor derangements involving the peripheral nerves.[5,32] In practically all instances these manifestations can be rapidly alleviated by providing the daily requirements.

An international unit of thiamin potency has been established as the physiological effect of 0.003 mg. of crystalline thiamin. At the present time, the daily requirement of the adult is presumed to be in the neighborhood of 30 international units.

Therapeutic indications for the use of thiamin with particular reference to surgery include:[5,32]

1. Preoperative preparation of alcoholic individuals. Chronically alcoholic cases are predisposed to serious diseases associated with vitamin B deficiency, particularly thiamin deficiency. The preoperative administration of adequate quantities of thiamin should be reckoned to correct any possible deficiency before surgery is undertaken.

2. Postoperative cases in which anorexia materially interferes with normal recovery. In these instances in which there is persistent lack of appetite, particularly if there is the possibility of a B deficiency, excellent improvement in appetite is frequently obtained by the daily administration of relatively large amounts of thiamin.

3. In instances in which surgery of the gastrointestinal tract is contemplated. The rationale of thiamin administration in these instances arises from an observation that thiamin deficiency produces various degrees of atony of the intestinal musculature with consequent impairment of function. Adequate thiamin administration is, therefore, indicated to preserve intestinal tone and function postoperatively.

4. In thyrotoxicosis when incident to the high basal metabolic rate there is increased destruction of thiamin. Due to the rapid rate of destruction, thiamin deficiency may develop even when intake and absorption are adequate. For the normal state it is, therefore, advisable to provide for this contingency preoperatively by giving increased amounts preoperatively.

RIBOFLAVIN

The limited application of the riboflavin component of vitamin B complex in relation to surgical therapy warrants only the briefest discussion. Riboflavin is a yellow crystalline water soluble substance formed from the organic union of the chromophoric nucleus flavin in a molecule with the sugar a-ribose. Its precise physiological function is obscure although it is presumed to be a component of all yellow oxidation enzymes and, therefore, an essential in cellular respiration. Deficiency of riboflavin produces a specific type of cheilosis and ocular manifestations including impaired vision, increased pigmentation of the iris and keratitis.[33,34]

Daily requirements are adequately provided for in an ordinary balance diet or by the administration of 2 or 3 mg. In instances of deficiency daily dosage up to 50 mg. for five days is advocated.

Specific indications for its use in relation to surgery are lacking.

NICOTINIC ACID

Nicotinic acid is a white water soluble crystalline powder of basic pyrimidine structure. It is chemically allied to nicotine but has no pharmacological similarity to the latter substance. It is almost quantitatively absorbed from the gastrointestinal trace and other sites of administration. In human beings, in therapeutic doses, nicotinic acid rather promptly results in a vasodilatation.[35] There may be a marked blushing with mild burning or itching sensations. In order to obviate this unpleasant sensation the nicotinic acid amide is now more widely used therapeutically. In greatly increased quantities it is capable of producing toxic symptoms, at least in experimental animals.

Like the two former B complex substances it is apparently an essential constituent in the enzymatic control of cellular oxidation. Deficiency results in the pellagra syndrome with symptoms reflected in the skin by cutaneous eruptions, pigmentation and ulceration; in the gastrointestinal tract by ulceration of the buccal mucosa, nausea, vomiting and diarrhea; and in the nervous system by vertigo, insomnia and peripheral nerve disorders.[35]

Specific therapeutic indications for the administration of nicotinic acid which are definitely related to surgery are difficult to ennumerate. The chief indication for its use is in surgical cases in which the deficiency is diagnosed in preparation for surgery. It has been suggested as routine preoperative therapy in conjunction with thiamin and if this suggestion is accepted indications for its use are the same. This may also be said of riboflavin.

Nicotinic acid is employed therapeutically as nicotinic acid, as the amide or as the sodium salt. Variations exist in individual daily requirements being variously given as from 100 to 1,000 mg. When no deficiency is clinically apparent, a dosage of 50 to 100 mg. three times daily should be adequate preoperative dosage. In actual deficiency states this dosage can be increased four or five times without harm. Nicotinic acid or its derivatives may be given subcutaneously or intravenously when oral administration is not feasible. Occasional reactions are encountered in increased dosage consisting of blushing, pruritis and mild fever, rarely of abdominal pain, nausea and vomiting. These latter effects are transient and subside in a short time.

VITAMIN C

Although the clinical symptoms associated with advanced vitamin C deficiency have been a familiar clinical syndrome for many years, its specific relation to the process of body repair of injured tissue has only recently been appreciated. Before considering this important function, a brief review of the general aspects of the vitamin C factor is appropriate.

Vitamin C is a crystalline, water soluble, optically active substance of acid form, to which the name of ascorbic acid and cevitamic acid has been given. It is isolated in greatest quantities from fruits of the citrus group and has been synthesized.

It is a strong reducing agent and is readily destroyed by oxidizing substances. Under usual circumstances there is almost complete absorption of vitamin C from the gastrointestinal tract, after which increased amounts may be directly measured in the blood. Quantitative determination of the blood level of vitamin C measured as ascorbic acid is the best single indication of its presence in adequate quantity. In the human being between 0.5 and 1.0 mg. represents the normal state while values below and above these values approach deficiency or saturation. Levels below 0.15 are invariably associated with outwardly manifest clinical signs of deficiency. It has been conclusively shown that when the blood and tissues become saturated with ascorbic acid that the excess is largely excreted through the kidney by a threshold mechanism which is in the neighborhood of 1.5 mg. per cent. Administration of the substance after a period of deprivation results in its rapid removal from the blood by the tissues. This tends to keep the blood level low. Because ascorbic acid is destroyed in the body by oxidation, constant replenishments are essential. Daily requirements in the human are in the neighborhood of from 20 to 50 mg. in children and 50 to 100 mg. in adults. In certain disease conditions the vitamin C requirements are increased above normal due to an increased rate of destruction. This is particularly evident in certain infections[37] diseases, pregnancy, cancer, peptic ulcer and hyperthyroidism. The deficiency effects concerned with vitamin C are largely a result of the changes which are induced in certain mesenchymal cells which form the supporting structure of various body tissues.[38] In deficiency states the collagen intercellular substance of all fibrous tissue derivatives including that of the blood vessels, skin, cartilage, bone and teeth is altered so that there is a marked derangement in structure and function. This fundamental effect is presumed to be the underlying cause of most of the pathological changes observed in scurvy and is particularly evident from controlled observations on body repair of traumatized tissue and wound healing. Since the latter are of ultimate surgical concern they will receive further consideration.

A wound, however inflicted, represents a solution in the continuity of tissue, and the same essential process of repair is followed in all instances beginning almost immediately after injury. At first cellular and fluid elements pass out of the blood into the injured area, with the formation of fibrinous strands which upon contracture produce a protective clot. Necrotic tissue is broken down by autolysis and carried away by phagocytic cellular elements leaving a framework of crystallized fibrin uniting the surrounding tissue. Connective tissue cells in the adjacent tissue begin a proliferative stage extending their process out along the fibrinous strands digesting and replacing the latter as they grow. With the production of increased amounts of collagenous intercellular substance new fibrous tissue is formed into which the surrounding blood vessels begin to grow. Ultimately, there results a complete replacement of the tissue defect with soft highly vascular fibrous scar. Gradually this scar undergoes contraction largely by reason of the conversion or alteration of its precollagenous elements into collagen. Surface epithelium grows in from the edges to cover the lesion and the process of repair is complete. It is this progressive conversion of the precollagen substance of the scar to collagen which gives the great tensile strength characteristic of old normal scar tissue. When there is insufficient vitamin C, the healing of the wound as a whole is profoundly disturbed. Collagen is not formed from its precursor substances and the intracellular material does not mature so that normal tensile strength is not acquired. In addition the proliferating connective tissue cells remain in a state of immaturity. Moreover it has been demonstrated that the collagen of old and normally healed wounds is reconverted to precollagen if C deficiency supervenes.[38]

It is obvious, therefore that vitamin C in adequate amounts is a prime requisite of normal wound healing.[38–41]

Other nutritional factors in wound healing must be considered in conjunction with vitamin C, particularly the indispensability of adequate serum protein. In conditions of severe hypoproteinemia the resulting edema may dangerously delay the healing process.[42,43] This is of particular interest in the surgery of the gastrointestinal tract where the procedures of resection and anastomosis may become extremely hazardous because of poor healing even when the actual technic is entirely above question.

From the foregoing considerations it will be seen that the indications for investigation of the vitamin C status and institution of adequate therapy are particularly urgent in: (1) All instances in which the signs of deficiency are grossly apparent;[37] (2) all cases in which major surgical procedures are anticipated or have already been carried out;[37,44,45] (3) all cases with a history of chronic gastrointestinal tract disease;[46] (4) cases of delayed healing for which there is no apparent cause in surgical or traumatic wounds;[45] (5) instances in which dietary restrictions have been voluntarily imposed as a therapeutic measure for various other diseases;[46] and (6) disease in which there is abnormally rapid depletion of vitamin C body content due to increased rate of breakdown.[37]

When no deficiency exists a daily intake of 120 mg. of ascorbic acid is adequate to provide for the highest normal requirements. Since there are no toxic consequences in giving excessive dosage, larger amounts may be given routinely. In instances of infectious diseases, neoplastic disease, chronic gastrointestinal ailments and hyperthyroidism the daily maintenance dose should be correspondingly increased. In marked deficiency states a dosage of 1,000 mg. of ascorbic acid daily for three days will produce saturation and 100 to 120 mg. is usually enough to maintain it. When such gross deficiency exists, it is expedient where possible to examine the ascorbic blood level values at intervals as a check on the adequacy of treatment.

SUMMARY

Vitamin therapy is of considerable importance in the pre- and postoperative care of surgical patients.

Those of particular surgical interest are vitamins K, certain component factors of vitamin B and vitamin C because they are specifically related to the problem of nutrition and to body functions essential to normal tissue repair after surgery.

Certain aspects of the general subject of vitamin therapy have been considered.

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THE SCIATIC SYNDROME

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LOW backache accompanied by sciatic nerve radiation of the pain, with or without actual evidence of nerve irritation, may be termed the sciatic syndrome. Since sciatic neuritis is a manifestation of some systemic diseases, such as diabetes, gout, etc., it does not fall into this category. The condition is a symptom complex, and not a diagnosis, which may be produced by any one of a number of lesions of the lower back and pelvis. The scope of this paper shall be limited to a discussion of these various lesions, and some of the differential characteristics of each will be given. No attempt will be made to enter into the treatment except for certain measures which may be of diagnostic aid. It is absolutely necessary to arrive at a fairly accurate diagnosis before rational treatment can be instituted. Treatment which is not based on a knowledge of the underlying cause, at best, cannot be adequate and most likely will be ineffective, even harmful in many instances. On the other hand, once an accurate diagnosis has been made the prognosis for an effective cure can be expected.

In order to approach intelligently the problem, one has to possess a basic knowledge of the regional anatomy and the rôle played by the various structures in the production of the disability. The symptoms and various signs accompanying them are of definite significance and one must be able to interpretate these in terms of the pathological condition involved in order to arrive at the basic cause. The failure to recognize or the ignoring of these important facts will be followed by failures in most instances.

ANATOMIC AND PATHOLOGIC CONSIDERATIONS

The change of the human being from a quadruped to a biped and the frequent presence of defects in the development of the low back structures have subjected the muscles, ligaments and joints of this area to strains and other lesions with radiated pain. It is true that pain in the lower back occurs more frequently without sciatic radiation of the pain than with it. However, any type of lesion in the area may produce the typical syndrome. A review of the anatomy, particularly the overlapping of the nerve supply, adequately reveals how this may occur.

The sciatic nerve arises from the fourth and fifth lumbar and the first, second and third sacral roots. Since the spinal cord ends at about the level of the first lumbar vertebra, the nerves are given off considerably higher in the canal than the point at which they make their exit. The anterior and posterior roots pierce the dura separately but join immediately afterward to compose the whole nerve root. Just after these nerves leave the canal they divide into anterior and posterior divisions. The posterior division supplies branches to the articulations, ligaments, muscles and their bony attachments. The anterior division gives off branches to supply some articulations, muscles and structures in the pelvis, but the main trunks make up the sciatic nerve. A lesion of any of the structures supplied by branches of the nerves, before joining to form the sciatic trunk, may produce reflex pain,[9] while pressure on the roots may produce direct nerve irritation. The former group is rarely accompanied by any objective findings.

Each of the vertebrae is jointed together by an intervertebral disc and two articular facets. The intervertebral disc is made of cartilage plates which are attached to the surfaces of the vertebral bodies above and below by a thin layer of calcified cartilage. Lying in the plates posteriorly is the nucleus pulposa. The annulus fibrosa sur-

rounds these structures and is firmly attached to both bone and epiphyseal plates by means of the Sharpey fibers. The nucleus pulposa lies beneath it, forming the anterior wall of the intervertebral portion of the spinal canal. Any condition resulting in the rupture or weakening of the annulus fibrosa may be followed by a displacement of the nucleus pulposa into the canal. The displacement is usually posterolateral on one side or the other. The displaced mass produces direct nerve pressure over the nerve root as the latter passes toward the intervertebral foramen.

The articular facets are true joints consisting of articular cartilage, synovial lining and supporting ligaments. Motion takes place in the joints as it does in all those of the diarthrodial type. These are subject to strains and other types of disturbances which may be followed by a synovitis and periarticular swelling. Since they form the posterior wall of the intervetebral foramen they lie in direct contact with the nerve root as it makes its exit from the canal. By reason of this location, pressure may be produced directly on the nerve. This occurs most frequently in the lumbosacral joint where the foramen is the smallest and the fifth lumbar nerve the largest. Muscle decompensation and ligamenture strain, resulting in an increase in the lordosis at this joint, further narrow the foramen and often produce sciatic pain by direct irritation of the nerve.[1,2]

The nerve supply to the capsule and supporting ligaments of the above joints and the sacroiliac joints is such that lesions affecting them may produce sciatic pain. This is also true of the ligamentous attachment of the muscles and the muscles themselves in this area.[3,4,5]

ETIOLOGY

The sciatic pain which accompanies low backache can be explained on one of two groups of causes, namely, those of reflex origin and those in which there is direct nerve irritation. Injury or disease of the structures that are supplied by the nerves which arise from the same trunks that compose the sciatic nerve may produce a reflex sciatic pain, while lesions adjacent to the nerve roots may produce direct irritation of the nerves. The former rarely disturbs the function of the nerve while the latter often is accompanied by a decrease in reflexes, localized sensory changes and at times some muscle weakness. Therefore, any classification based on these groups would be anatomic and not etiologic. The various causes of low backache with radiation of the sciatic pain have been tabulated for the purpose of aiding one in arising at a diagnosis. Since no standard classification has been offered, the one here is based on the interpretation of findings by a few individuals:[6,7]

I. Congenital Anomalies
   - A. Anomalies of the facets and pedicles
     1. Malformation and malposition of the facets
     2. Separation of the neural arch (may produce spondylolisthesis)
   - B. Absence or narrowing of the lumbosacral disc
   - C. Sacralization of the fifth lumbar vertebra or lumbarization of the first sacral vertebra
   - D. Spina bifida

II. Trauma
   - A. Strains
     1. Acute
     2. Chronic
        - a. Postural
        - b. Repeated injury
   - B. Old fractures
     1. Body
     2. Pedicles
     3. Facets
     4. Lamina
   - C. Localized arthritis
     1. Bodies of the vertebrae
     2. Facets
   - D. Injuries to the intervertebral discs
     1. Narrowing of the intervertebral disc

2. Protrusion of the nucleus pulposa into the spinal canal
3. Avulsion of the disc

E. Hypertrophy of the ligamentum flavum

III. Inflammatory Lesions
A. Arthritis
1. Rheumatoid
2. Spondylitis deformans
B. Fibrositis
C. Specific infections

IV. Metabolic Lesions
A. Osteoarthritis
B. Gout
C. Osteoporosis
1. Senile
2. Disuse
3. Hyperparathyroid disease

V. Neoplasm
A. Benign
B. Malignant
1. Primary
2. Metastatic

VI. Primary Cord Lesions
A. Tumors
B. Degenerative diseases

VII. Malingery

HISTORY AND EXAMINATION

A carefully taken and thorough history is most essential to gaining an insight into the complaints of low backache with sciatic pain. This requires a careful follow-up of all the leads one can obtain while questioning the patient. Frequently the patient cannot recall anything concerning the original onset of the trouble, but by a careful checking and rechecking of all the answers one can bring out incidents which will recall the basic cause. In most cases, this one point may clarify the entire problem of diagnosis. The history of previous treatment is often helpful in solving the problem, as well as avoiding a repetition of some therapeutic measures. So often one may be prone to pass off the problem as a backache or sciatica when in reality these are only symptoms and not a diagnosis. This often results in inadequate or incorrect treatment. If, on the other hand, a thorough history has been obtained, the examiner has already secured many of the data necessary to solve the problem.

In order to give the examiner a definite approach to each case, a skeletal history outline has been worked out.[6] This outline gives only the basic points to be analysed. Each subhead will have to be followed out separately. Each and everyone of the headings and subheadings are important. The overlooking of any one of these may mean the failure to obtain the correct insight into the specific case that is being studied. For the purpose of clarification the sciatic pain is listed under a separate heading in this outline, but the relation of this pain to the backache must always be ascertained.

A. Age and occupation
B. Pain
1. Factors accompanying and preceding onset
2. Mode of onset
3. Duration
4. Location
a. Localized
b. Diffuse
c. Radiation
5. Type and character of pain
a. Continuous
b. Intermittent
c. Aching, boring, throbbing or soreness
6. Effect of rest and activity
7. Relation to standing
8. Time
a. Continuous
b. Morning
c. End of day
d. Night
C. Soreness and stiffness (if present, is it worse on arising or after activity)
D. Feet symptoms
E. Sciatic Pain
1. Relation to the backache
2. Continuous
3. Intermittent or recurrent
a. Night or when lying on back with lower extremities in extension
b. Standing or during activity

c. Relieved by rest
d. Effect of coughing or sneezing
4. Tiring or difficulty when walking
5. Bowel and bladder disturbances

F. Symptoms relative to remote structures and their relation to the backache

G. Previous therapy

A complete routine examination should be carried out on every patient. Before beginning the examination, the patient must be completely disrobed, including the removal of the shoes and stockings. The female patient may be draped but the entire back and lower extremities should be exposed. It is most important for a complete general examination to precede that of the back. At this time special attention is given to the teeth, tonsils, lower gastrointestinal tract, genital organs and urinary tract. Infection in these structures may be the cause of an arthritis, or a minor disturbance in them may produce a reflex type of pain. However, the latter is a rare occurrence. Tumors in the pelvis, bladder and rectal area are important in that they may produce the same type of pain or they may involve the spine or nerve by extension. The actual physical study of the back begins as soon as the patient enters the office. By doing this one can often obtain valuable information when the patient is not aware that he is being observed. This is particularly true in the malingerer.

Upon beginning the examination the patient is asked to walk, stoop, walk on the heels and the toes, hop and squat, during which time one notes the disturbance in posture, gait and impaired muscle function. Following this the back and lower extremities should be examined in the standing, sitting and both the prone and supine positions. The latter phase of the examination should be carried out on a firm table that is long enough for the patient to stretch out comfortably. The following general outline has been found to be of aid in observing and tabulating the findings:

1. General posture
2. Gait
3. Contour of the back
   a. Normal
   b. Kyphosis
   c. Lordosis
   d. Lateral curvature
      (1) Scoliosis
      (2) Lateral deviation or what is generally termed "sciatic scoliosis"
4. General protection of the back in moving
5. Tenderness
   a. Location
   b. Localized or diffused
   c. Degree of severity
   d. Whether or not eliciting it reproduces back or sciatic pain
6. Muscle spasm
7. Spinal motions
   a. Guarded
   b. Actual restrictions
   c. Does eliciting the motion reproduce the back or referred pain.
8. Gaenslen's test for sacroiliac disease
9. Patrick's sign
10. Ober's test for shortening or spasm of the tensor fascia lata[3]
11. Straight leg raising test
12. Prone trust test[9]
13. Trigger point test[5]
14. Evidence of sciatic nerve involvement
    a. Tenderness
    b. Positive Lasegue's sign
    c. Decreased or absence of the Achilles tendon response
    d. Muscle weakness and atrophy
    e. Sensory changes

The major symptoms and physical findings in the sciatic syndrome are so frequently observed that a brief elaboration of them has been thought to be of some value.

Pain. Pain in the lower portion of the back is present in all cases with radiation to the buttock, posterior thigh and then down the course of the peroneal nerve or it passes down the outer side of the leg and to the foot. The back pain may be a constant dull ache, a sudden catch upon any motion or a

combination of the two. The sciatic pain may be continuous, intermittent or it may be present only on sudden motion and lasting for just a short period.

Posture. The back may be straight with good motion in all directions; however, forward bending is limited in most instances because it produces some tension on the nerve trunks. There may be an increase in the forward lumbar curve producing the so-called "sway back." This is seen more frequently in women with relaxed and weak muscles. There may be a list to one side or the other, the so-called sciatic scoliosis. More often the list is away from the painful side and is accompanied by slight flexion at the hips. When present, attempts to bend to the opposite side or to straighten up increases both the back and referred pain.

Signs and Tests. 1. *Prone Thrust Sign.*[9] The patient lies prone, raises himself on his hands with the elbows extended, dropping the pelvis toward the table as far as possible. This produces hyperextension of the spine with the strain centered in the lumbosacral area.

2. *Gaenslen's Test.*[8] The test is elicited with the patient in the supine position and the painful side to the edge of the table. The thigh of the well extremity is forcibly flexed on the abdomen to fix the lumbosacral joint. The other extremity is then dropped over the side of the table and slight forced hyperextension at the hip is carried out. The reproduction of the pain in the sacroiliac area suggests involvement of this joint. It is believed that this is one of the most reliable tests.

3. *Patrick's Sign.* The forced abduction of the flexed thigh produces pain in the sacroiliac region. The test allows some rotation at the lumbosacral joint; thus its value as a diagnostic test of sacroiliac joint involvement is questioned.

4. *Ober's Test.* Ober's test is indicative of shortening of the iliotibial band. It consists of an attempt to adduct the abducted extended thigh with the knee flexed and the extremity supported only by the foot. The test is carried out with the patient lying on the side. The failure of the knee to drop down to the table indicates some shortening of the muscle.

5. *Trigger Point Test of Steindler.* The presence of a localized point of tenderness, which when injected with novocain produces relief of the radiated and local pain, indicates a local ligamentous or muscle strain.

6. *Straight Leg Raising Test.* The lower extremity is flexed at the hip with the knee extended, producing pain in the hamstring and lumbosacral area. The extension of the lordosed lumbosacral joint by the tense hamstring muscles offers the best explanation for the pain.

7. *Lasègue's Test.* The thigh is flexed to right angles. The leg is extended on the thigh until pain begins in the buttock or posterior thigh region. No further movement of the thigh or leg is carried out, but the foot is then dorsoflexed. The latter phase of the test increases the tension on the sciatic nerve without moving or putting any further strain on the hip or lumbosacral spine. If the pain is increased in the course of the sciatic nerve, the test is considered positive. This test should be definitely differentiated from the so-called straight leg raising test because it only places tension on the sciatic nerve while the latter also increases the strain on the low back structures.

8. *Muscle weakness, sensory changes and decreased reflexes will be found localized to the dermatomes which have been involved.* The presence of these signs indicate pressure on the sciatic nerve or the roots from which it arises.

9. *Tenderness in the lower back is of importance because of the manner in which it has to be elicited.* When jarring is required to elicit it the source is usually in the bone or joint structures. If pain is produced in the sciatic nerve area by applying deep pressure to the involved side of the suspected vertebra, one suspects a ruptured nucleus pulposa. Tenderness over the ligamentous attachments about the poste-

rior lower back area is significant if the pain is relieved by a local infiltration with novocain.

Special Examinations. 1. *Roentgenologic Examinations.* The physical examination should be followed by routine stereo-anteroposterior and lateral *x*-ray films of the lower back. If one suspects a lesion of the facets, the views should be supplemented by films in both oblique positions. In the *x*-ray one has a valuable aid in the diagnosis of many lesions, but if one depends on it for the diagnosis he will encounter many pitfalls. The interpretation of the findings and the correlation of them with the clinical findings are absolutely essential and require a knowledge of the variations in the skeletal structure of the lower back as well as the pathologic bone changes which may occur in this area. Ghormley[11] has tabulated these changes in detail.

2. *Spinal Puncture.* A spinal puncture should be performed on every patient in whom the clinical findings indicate a lesion inside the spinal canal and also on those patients in whom one suspects such a lesion when no other cause can be found. The puncture should be performed as low in the canal as possible, usually between the fifth lumbar and the first sacral vertebrae. A partial block in a lesion above this level can often be demonstrated. The removal of the fluid from the immediate area of the lesion, which is facilitated by the low puncture, is useful in showing the total protein elevation. This is explained on the basis that the increased protein in the spinal fluid is dependent upon circulatory disturbances and they are greatest in the immediate vicinity of the lesion.[11]

3. *Spinogram.* The instillation of any material into the spinal canal should be reserved solely for the purpose of localizing the lesion. Therefore, it must always be preceded by a positive clinical diagnosis. The technic now used by many surgeons is that of instilling air into the canal, because many of them believe that the presence of iodized oil in the canal produces some arachnoid irritation. Bradford and Spurling believe that this is not the case. They are of the opinion that the injection of a small quantity (2 cm.) of undeteriorated oil in the canal does not produce any irritation. The oil is a much more satisfactory aid to the diagnosis. The difficulty often encountered in visualizing the canal with air, together with the fact that it is more likely to be used in cases in which there are no definite indications for a spinogram, are logical reasons for not using it.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of low back pain accompanied by radiation of the pain to the distribution of the sciatic nerve presents many complex problems. Some lesions produce very definite and characteristic findings, while in others the symptoms and signs are so vague that a solution cannot be made even by the most experienced investigator. With the full realization of these facts, an attempt has been made to list the characteristics of the various groups of lesions.

Congenital Anomalies. Much significance has been placed on congenital anomalies as a cause of backache. When present they are evident enough to cause very little difficulty in arriving at a diagnosis. The roentgenologic findings are definite in this type of lesion. The problem is to be sure that such a lesion, even though present, is the cause of the symptoms.

Strains. The postural strains are characterized in the early stages by an aching and tired feeling while standing which is relieved by activity, gradually progressing until the pain is constant and accompanied by sciatic radiation. There is a lumbar lordosis with soft, flabby muscles while the majority of other findings are absent. Often, the etiologic factor, particularly in women, will be ill-fitting shoes. Thus, the feet are of sufficient importance to be always included in the examination. Strains resulting from injury usually are aggravated by activity. The pain is practically always localized to the structures under

strain. Active contraction of the involved muscles or passive stretching of the ligaments and ligamentous attachments will reproduce the pain. The injection of novocain into the localized area will relieve the referred as well as the local pain.

Traumatic Arthritis and Old Fractures. A history that injury initiated the symptoms can be obtained in nearly all cases. The pain is localized and it is aggravated by activity. When sciatic nerve radiation of the pain is present, it may signify that bony or scar tissue impingement of the nerve root or the actual compression of herniated nucleus pulposa is the cause.

Herniation of the Nucleus Pulposa. Trauma to the back, even though mild, followed by persistent or recurrent pain in the lower part of the back with sciatic radiation of the pain which increases with activity, suggest a herniation of the nucleus pulposa. The diagnosis can be made when this picture is accompanied by a diminished or absent Achilles tendon response, sensory changes and an increased concentration of protein in the cerebral spinal fluid. The above findings are indications for performing a spinogram to localize the lesion.

Hypertrophy of the Ligamentum Flavum. The clinical pictures produced by a hypertrophy of the ligamentum flavum and herniation of the nucleus pulposa are identical. The spinogram may show the posterior rather than the anterolateral defect, but since they occur together very frequently the differential diagnosis cannot be made before exploration.

Tumors of the Canda Equina. Cord tumors in the lumbar region often simulate herniation of the nucleus pulposa. However, the pain is usually persistent and progressive, increased at night or during rest and often relieved by activity. The findings resulting from nerve compression are more widespread, for example, muscle weakness, atrophy, sensory changes, reflex changes, etc.

Inflammatory Lesions. Pain in the back characterized by an onset of soreness and stiffness on arising in the morning which subside with activity only to recur with rest is characteristic of an inflammatory process. The condition may involve the joints, ligamentous structures and muscles of the back. The tissues involved will determine the location of the pain. Muscle spasm and rigidity accompany the pain. Limitation of chest expansion often gives a clue to the solution of the problem. The condition will often be precipitated by an acute upper respiratory or other regional infection. Sciatic radiation of the pain may be present and exhibit all the characteristics of compression. The characteristic history and findings will differentiate the conditions. Lesions resulting from specific types of infection can usually be recognized by the roentgenogram and by positive laboratory data. Tuberculosis produces a destructive lesion, accompanied by a paravertebral abscess which may produce psoas muscle irritation. Malta fever presents a picture of a destructive arthritis usually without a paravertebral shadow. The agglutination or intradermal test will indicate the presence or absence of the infection.

Metabolic Lesions: Osteoarthritis. Osteoarthritis rarely will produce sciatic pain by means of direct irritation of the root by a large spur. The symptoms are present on activity and subside with rest. Rarely are any objective nerve findings present. The presence of a large spur corresponding to the root pain in the absence of any other cause makes the diagnosis.

Gout. Recently attention has been focused on gout. The picture may be that of an acute onset subsiding in a few days with periodic recurrence, or it may simulate the usual infection group. The elevation of the blood uric acid and the response to the diagnostotherapeutic test indicate its presence.

Osteoporosis. Osteoporosis of the spine nearly always produces backache. If the softening of the bone is sufficient to allow some compression of the vertebral bodies, there will be some disturbance of the articular facets and possibly some narrow-

ing of the intervertebral foramen. Either of these may produce sciatic pain. The pain will be increased by activity and relieved by rest. There is marked diffuse tenderness on jarring and back motion is limited. The condition is found most frequently in debilitated and elderly persons; however, those cases which are manifestations of hyperparathyroid disease may be found in young people. The diagnosis of the whole group can be made by the roentgenogram while the blood chemistry is of primary importance in differentiating the hyperparathyroid group.

Neoplasms. The benign tumors will cause little concern because they can always be diagnosed by the roentgenogram. Malignant tumors, on the other hand, may confuse one, especially in the early stages because at this time there may not be any radiologic evidence of changes in the bone. When persistent night pain is present for which no skeletal origin can be found, when the neurologic examination is negative and when the condition does not respond to the usual forms of treatment, one must consider the presence of a malignant neoplasm. A recheck of the patient after a few months will often reveal the nature of the lesion to be a malignant tumor.

Remote Causes. Minor disturbances of the pelvic organs in both the male and female rarely cause back pain. However, they are the cause just frequently enough so that one has to consider them when arriving at a diagnosis. Pain with its origin in the pelvis is most often referred to the center of the sacrum and sacroiliac joints, radiating to the distribution of the sciatic nerve. Tumors in the pelvis may, by means of their size and location, produce pressure directly on the sacral plexus. When this occurs they may simulate any of the various causes of the syndrome. The diagnosis can be made by a careful pelvic and rectal examination.

Malingery. The malingerer frequently presents a diagnostic problem. Careful observation during the whole course of the examination will often expose these individuals. Their lack of anatomic knowledge makes it nearly impossible for them to fake an organic lesion. Almost invariably the findings are far more extensive than could be produced by any disease process. The symptoms and findings will not follow the pattern of any organic lesion.

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LYMPHADENOPATHY OF THE NECK

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IN considering lymphadenopathy of the neck it is necessary to take into account various conditions which may cause or be mistaken for enlargement of these lymph-nodes. These conditions have been listed in Table 1 and also will be considered under separate headings.

TABLE 1
CONDITIONS SIMULATING OR CAUSING ENLARGEMENT OF THE LYMPH NODES OF THE NECK

- Cystic tumors
  - Branchial cysts
  - Thyroglossal cysts
  - Cystic hygroma
  - Dermoid cyst
- Vascular lesions
  - Aneurysm
  - Angioma
- Inflammatory lesions
  - Syphilis
  - Tuberculosis
  - Acute suppuration
  - Woody phlegmon
  - Actinomycosis
  - Blastomycosis
  - Infectious mononucleosis
- Solid tumors
  - Benign tumors
    - Lipoma
    - Mikulicz's disease
    - Adenoma of thyroid gland
  - Malignant tumors
    - Metastatic carcinoma
    - Carcinoma of thyroid gland
    - Adenocarcinoma of submaxillary gland
    - Tumor of carotid body
    - Lymphoma
      - Hodgkin's disease
      - Lymphosarcoma
      - Lymphatic leukemia
      - Leukemic reticulo-endotheliosis

LIPOMA

A lipoma usually is rather soft and it is poorly defined. Often, on careful palpation, the lobules of the tumor can be felt. Usually, there is a history that the mass has been present without material change for a long time, often for years.

MIKULICZ'S DISEASE

Mikulicz's disease is a process of lymphocytic replacement of the glandular structure of the salivary and lachrimal glands. This causes the glands to become enlarged and firm. If the submaxillary glands in the neck are enlarged and firm and if the parotid and lachrimal glands present evidence of similar induration and enlargement, the presence of Mikulicz's disease must be considered.

ADENOMA OF THE THYROID GLAND

Adenoma of the thyroid gland ordinarily need cause little confusion because it usually is in a lobe of the thyroid. Occasionally, however, an adenoma may be found in an unusual situation and may closely simulate an enlarged lymph-node. The most common atypical site is in the region overlying the thyroid cartilage. This is above the isthmus and is in the region where metastasis of carcinoma of the larynx may be found.

PARAFFINOMA

A rare cause of a nodule in the neck is paraffinoma. Paraffin is sometimes injected subcutaneously for the correction of facial deformities, particularly wrinkles and depressions. This material may migrate down into the neck and lead to the formation of firm fibrous nodules. On palpation, these are likely to resemble none of the conditions commonly encountered and this fact may serve as a clue so that careful questioning may bring out the fact that paraffin has been injected for cosmetic purposes.

BRANCHIAL CYSTS

A branchial cyst usually is a perfectly smooth globular mass situated in the

midcervical region just anterior to the sternomastoid muscle. Its cystic character usually can be made out on palpation but sometimes it is so tense that it feels like a firm lymph-node. If the cyst has drained spontaneously or has been drained surgically, there may be a draining sinus leading to an indurated region. The condition then may resemble tuberculous adenitis. Careful palpation will reveal a cord-like structure leading from the sinus to the region of induration. This is not characteristic of any other condition (except for some thyroglossal cysts).

THYROGLOSSAL CYSTS

A thyroglossal cyst differs from a branchial cyst chiefly in situation. It is found in the median line overlying the thyrohyoid membrane. If intact, it has the same characteristics on palpation as a branchial cyst. There may be a draining sinus (sometimes opening as low as the suprasternal notch) and a palpable cord-like structure leading to the middle of the hyoid bone.

CYSTIC HYGROMA

Cystic hygroma is a tumor made up of multiple cysts which have thin walls and are filled with lymph. If not infected, it is very soft and easily compressible. If it is infected, it may be very tense, the overlying skin is usually red and the patient is likely to appear acutely ill because of the ready absorption of toxic material from the thin-walled structures making up the tumor.

DERMOID CYSTS

Dermoid cysts occasionally occur in the floor of the mouth and produce a mass visible and palpable in the submaxillary region. Characteristic features are the very slow progression and the fact that on bimanual palpation a distinctly doughy sensation is noted.

ANEURYSM

An aneurysm of one of the large vessels of the neck is a rare condition but should present no great diagnostic difficulties. It is a fairly tense but compressible pulsating tumor over which a bruit can be heard. In patients, who are thin and elderly, the carotid artery just below its bifurcation may be bulbous or slightly tortuous. This finding is sometimes mistaken for aneurysm or even for an enlarged lymph-node.

ANGIOMA

An angioma of the neck which is deeply situated and does not involve the overlying skin will not produce the discoloration usually characteristic of angioma. There are no symptoms unless the tumor is very large. On palpation, such a lesion is soft, easily compressible and is very poorly defined. No bruit is to be heard on auscultation over the lesion. Usually, the tumor becomes noticeably larger when the patient's head is placed in a dependent position. It may be difficult to differentiate between cystic hygroma and angioma. The former tends to be softer and more easily compressible than the latter.

SYPHILIS

Syphilis often is a cause for enlarged lymph-nodes in the neck. In primary syphilis, when the initial lesion is situated about the mouth or throat, enlarged lymph-nodes always are present in the neck. In cases of secondary syphilis they may be present in the neck as part of a general adenopathy. If there are any secondary lesions about the oral cavities, cervical adenopathy is almost certain to be present. In tertiary syphilis, unilateral cervical adenopathy is occasionally found. The enlarged gland usually is very firm and simulates a metastatic malignant lesion. If serologic tests are positive and if there are no other findings to account for enlarged lymph-nodes in the neck, a therapeutic test may be indicated. Unless the response is very prompt, some other explanation for the adenopathy must be sought irrespective of a positive serologic reaction.

TUBERCULOUS CERVICAL ADENITIS

Tuberculous cervical adenitis may be encountered at any age. The disease usually is unilateral and it may or may not be associated with draining sinuses. If sinuses are present, the history usually will reveal that the enlargement had occurred gradually and that softening and redness had been present for two weeks or more before the lymph-node drained spontaneously or was drained surgically. In cases in which the lymph-nodes do not break down, there usually is a history of enlargement and recession. The lymph-nodes tend to enlarge when the patient has a cold or some other condition which reduces his resistance. They tend to recede again as the general health improves. Tuberculous adenitis may be present in the neck when there is no demonstrable evidence of tuberculosis elsewhere in the body. Biopsy is necessary to make a positive diagnosis.

ACUTE SUPPURATIVE CERVICAL ADENITIS

Acute suppurative cervical adenitis usually causes little difficulty in the matter of diagnosis. Enlarged lymph-nodes in the neck, often unilaterally situated or larger on one side than on the other, appearing with or soon after an inflammatory process about the oropharynx, are likely to be the result of secondary inflammation. This condition is seen most frequently among children. The course of events usually removes any doubt as to the diagnosis. Resolution may take place but suppuration often occurs and drainage is necessary. Usually, one or the other course is evident within a few days.

WOODY PHLEGMON

Very rarely the acute suppurative adenitis becomes indolent and results in a condition known as woody phlegmon. In this condition the patient has an indurated mass in the submaxillary region or neck (or both), which may remain virtually unchanged for weeks or months. Usually, resolution without suppuration finally occurs.

ACTINOMYCOSIS

In actinomycosis a slowly progressive induration appears usually in the submaxillary or parotid region and very often it is associated with more or less ankylosis of the mandible. Frequently, there is a history of extraction of a tooth or of some other trauma within the mouth, usually a month or more before the appearance of the present symptoms and signs. In short, if the patient has what seems to be an inflammatory induration in the vicinity of the jaw, the duration of which is three weeks or more, and if there is an associated ankylosis of the mandible, the possibility of actinomycosis must be very seriously entertained. Microscopic examination of pus from a freshly opened pustule or abscess for the demonstration of the characteristic granules or mycelia is necessary to establish the diagnosis. It may be necessary to make repeated examinations before a positive smear is found. The organisms are cultured with difficulty.

BLASTOMYCOSIS

Blastomycosis is one of the rare causes of enlargement of the cervical lymph-nodes. Lesions of the mucous membrane, from which the disease may metastasize to the lymph-nodes, are granulomatous in appearance and have on their surface scattered tiny yellowish pustules. Skin lesions also are granulomatous and often crusted. If a specimen of a suspected lesion is removed for biopsy, the pathologist should be advised as to the suspicion because the characteristic double refractive organisms easily may be missed unless special care is used in searching for them.

METASTATIC MALIGNANT LESIONS

The physician must always be on the alert for metastatic malignant lesions as a cause of enlarged lymph-nodes in the neck. When found they usually are secondary to a primary lesion on the lip or in the mouth or throat. Metastatic involvement of the lymph-nodes by a malignant lesion produces a very characteristic sensation of

firmness when palpated so that the experienced physician can predict with a high degree of accuracy whether or not palpable lymph-nodes are the site of malignant metastasis.

Metastatic involvement of the cervical lymph-nodes by carcinoma of the lower lip, cheek or lower jaw is found most frequently in the submaxillary region. Metastasis of carcinoma of the tongue, hypopharynx or tonsillar region most frequently occurs deep in the carotid triangle of the neck. Carcinoma of the larynx most frequently metastasizes to lymph-nodes overlying the bifurcation of the carotid artery. Carcinoma of the nasopharynx may metastasize to the lymph-nodes in the carotid triangle of the neck or deep under the upper third of the sternomastoid muscle. When suspiciously firm lymph-nodes are found in any of these situations, careful search for a primary lesion must be made in all portions of the oral and nasal cavities. In the case of elderly patients, one must consider the possibility that metastatic involvement of a lymph-node in the neck may be secondary to "skin cancer." These lesions, particularly those in the preauricular region, sometimes are active squamous cell carcinomas and may metastasize. If no malignant lesion is found, the patient must be questioned carefully as to whether he has had treatment for a lesion in any of these regions. The patient may have had a sore on his lip several years previously, for which he had been given "light treatments" and he may be totally unaware that the lesion had been believed to be cancer and that he really had received roentgen therapy. Even in cases in which there is an obvious scar, the patient, when first questioned, may forget that he had ever had a lesion treated. One also must remember that there is a tendency among too many physicians to "try some *x*-ray treatment and see what happens." In some instances this therapy may result in the disappearance of the primary lesion without material effect on the metastatic involvement of the nodes. Biopsy of a suspected node may be the only means of reaching a decision.

Metastatic involvement of the lymph-nodes in the neck secondary to primary lesions in sites other than the oral cavities, such as the esophagus, stomach or breast, is likely to be found just above the clavicle. Suspicious lymph-nodes low in the neck should arouse suspicion and lead to investigation of these structures.

CARCINOMA OF THE THYROID GLAND

Carcinoma of the thyroid should cause no great diagnostic difficulties because of the situation of the characteristically firm mass which involves the gland and is fixed within it. If there is associated paralysis of a vocal cord it is almost certain that the suspected tumor is malignant.

A carcinoma of the thyroid gland may be very small yet give rise to metastatic involvement of the lymph-nodes. The involved lymph-nodes usually are found adjacent to and just posterior to the affected lobe of the thyroid gland. Usually the primary lesion can be felt on palpation but even if it is not palpable the site of the metastatic involvement of the lymph-nodes is sufficient ground for suspicion.

ADENOCARCINOMA OF THE SUBMAXILLARY GLAND

Adenocarcinoma of the submaxillary gland causes a tumor palpable in the submaxillary triangle. It may be impossible to distinguish it from metastatic involvement of a lymph-node although its location at the site of the submaxillary gland together with the fact that the gland itself cannot be palpated is cause for strong suspicion. It should be remembered, however, that when a stone obstructs the duct of the submaxillary gland infection occurs in the submaxillary gland and the infected gland feels stony hard on palpation. Consequently, stone in the duct should be ruled out as a possible cause for an enlarged, very firm submaxillary gland.

CAROTID BODY TUMOR

Carotid body tumor is a rare cause of swelling in the region of the bifurcation of the carotid artery. This tumor is very rarely susceptible to diagnosis preoperatively. It is attached in the bifurcation of the carotid artery and if a firm tumor is found in this situation which is movable to and fro but not movable up and down, one is entitled to suspect this lesion.

HODGKIN'S DISEASE

Lymphadenopathy associated with Hodgkin's disease most frequently occurs in the cervical region and early in the disease the involvement usually is unilateral. A solitary lymph-node or several nodes may be involved and the size of the nodes may vary. The nodes are firm and somewhat matted together. In general, early in the disease the patient is not particularly ill and usually consults a physician because of cervical adenopathy. In the early stage, an important point in the differential diagnosis of Hodgkin's disease, lymphatic leukemia and lymphosarcoma is the fact that in Hodgkin's disease the involvement of the lymph-nodes generally is unilateral. In leukemia and lymphosarcoma the involvement is symmetrical and splenomegaly usually is present. In our experience it is very rare to find an enlarged spleen early in Hodgkin's disease but in the late stages of the condition the spleen frequently is palpable. The diagnosis usually can be established by excision of a lymph-node and examination of the fixed tissue. There is no characteristic blood picture although late in the disease there usually is polymorphonuclear leukocytosis with associated monocytosis, but since this same picture may occur in many other conditions it can under no circumstances be considered as diagnostic.

LYMPHATIC LEUKEMIA

In lymphatic leukemia and leukemic reticulo-endotheliosis, including the Schilling type of monocytic leukemia, the lymph-nodes usually are discrete and involve both sides of the neck symmetrically. In the chronic forms, in addition to cervical adenopathy there usually is generalized, symmetrical, peripheral lymphadenopathy. The spleen usually is enlarged and in some instances may be of enormous size, out of all proportion to the degree of involvement of the peripheral lymph-nodes. The liver likewise is enlarged in most cases. In acute leukemia the lymph-nodes may enlarge rapidly and hemorrhage frequently occurs from the mucous membranes owing to a decrease in the number of blood platelets. Occasionally, in very acute leukemia, lymphadenopathy may be slight and the patient may consult his physician because of hemorrhagic manifestations or ulceration of the mucous membrane of the mouth. In addition, anemia of severe degree frequently accompanies acute leukemia and there is an associated fever. The diagnosis of leukemia usually is made by presence of an elevated leukocyte count and high percentage of immature lymphocytes. It must be borne in mind that in acute leukemia the total leukocyte count not infrequently may be less than 10,000, but on examination of a blood smear a high proportion of stem cells will be found. Leukemic reticulo-endotheliosis usually is readily identified by examination of a blood smear which reveals various stages of the development of reticulo-endothelial cells into lymphocytes. The typical reticulo-endothelial cell is a large cell with abundant cytoplasm and a round nucleus with one or more nucleoli. Chromatin strands are sharply differentiated from the parachromatin and usually extend transversely across the lesser diameter of the nucleus. Frequently, these chromatin strands are bent toward the central portion of the nucleus, giving the appearance of a longitudinal groove. In the stages of development from this cell to the lymphocyte, there is gradual rounding of the cell as a whole with concentration of chromatin and loss of nucleoli, but the grooving persists practically to the mature cell. In the acute forms

of the disease, many mature lymphocytes will be found. Removal of a lymph-node for examination of fixed tissue is necessary for the differential diagnosis of lymphosarcoma. The Schilling type of monocytic leukemia is quite similar to leukemic reticulo-endotheliosis. The diagnosis usually can be established by examination of a fixed blood smear where it is found that the reticulo-endothelial cell is developing into a monocyte rather than into a lymphocyte.

INFECTIOUS MONONUCLEOSIS

From the standpoint of adenopathy, infectious mononucleosis presents essentially the same clinical picture as leukemia or lymphosarcoma. The involved lymph-nodes are discrete and usually are bilateral and symmetrical. They frequently are quite tender to palpation and may increase in size at great rapidity. As a rule, the course of this disease is relatively mild but at times the clinical picture may simulate that of fulminating, acute leukemia. The diagnosis may be suspected from examination of leukocyte and differential counts and the absence of anemia. The leukocyte count rarely is higher than 40,000 or 50,000, and the percentage of lymphocytes in the differential count usually is less than 80, whereas in leukemia the percentage of lymphocytes is generally 90 or more. Examination of a blood smear is an essential diagnostic aid. Practically all lymphocytes are mature although the cells may be somewhat altered in size and may have a foamy type of cytoplasm with bizarre-shaped nucleoli, so-called leukocytoid forms of lymphocytes. Examination of these cells shows that they are not immature but are usually a variant of a perfectly mature lymphocyte. Occasionally, slight immaturity may be found but the general picture is that of a rather benign process. Infectious mononucleosis is almost invariably a benign process and treatment is palliative.

DIAGNOSIS IN CONVULSIONS*

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THE mystery and superstition that has clung to the problem of convulsive seizures since the earliest human records has largely been dispelled during the past two decades.

A new point of view and interest has arisen within the profession since the demonstration of the beneficial effects of convulsions in the treatment of mental disease. It is now possible to produce a convulsion at any time, in any person, by the simple snap of a switch (electric shock); or injection of the appropriate dose of convulsant drug (Metrazol).

Tests have been devised to bring out in the open, latent convulsant tendencies in the patient by means of hyperventilation (Rosette), insulin-hypoglycemia (Sakel); or by the forcing of liquids, after renal suppression with pitressin (Fremont-Smith, McQuarrie).

With this recent intimate knowledge regarding convulsions and the ability to initiate or control an attack, it is apparent that the *diagnosis of convulsive seizures* must be considered from an entirely different point of view than that formerly taught and held by the profession.

Reluctance on the part of the profession to render a prompt diagnosis and classification when convulsive seizures are encountered, stems partly from fear that the seizure may be associated with some form of "epilepsy," and partly from confusion as to the proper term to apply, when such a symptom appears for the first time in the history of the patient.

Lack of specific knowledge as to etiology, as well as disagreement amongst authorities as to terminology, prognosis and treatment, has permitted the public to retain the superstitions of the past, (often shared by the physician himself) which implied "hereditary taint," family disgrace and future mental deterioration if the diagnosis of epilepsy was made. The profession and public alike have been unwilling to face the truth in the early period of the onset of symptoms, when much could be done to control the attacks and prevent undesirable progress and sequelae.

Multiplicity of terms for the problem under consideration only adds to the uncertainty and confusion. The reader, if he prefers, may select (and with good authority) any one of the following: *The sacred disease* (Hippocrates), *disease of Hercules* (Greek), *falling disease* (Hebrew), *morbus comitialis* (Roman), *epilepsy* (Avicenna?), *grand mal* (French), *spectrum of epilepsy* (Foster Kennedy), *convulsive state* (Lennox and Cobb), *cerebral dysrhythmia* (Lennox), *convulsions, seizures, attacks* and *fits* (*Vulgar*).

Although agreement as to terminology has not been reached as yet by the authorities, it is well recognized today that *epilepsy is not a disease* and convulsions, whether single or recurrent, constitute merely a *symptom complex* inherent to the nervous system, not unlike so-called "reactions of defense," (vomiting, shivering, spasms of coughing, involuntary reactions to fright, pain, etc.).

As a *symptom complex*, it must be expected to have variable manifestations. It is obvious that one cannot "cure" a *symptom complex* as they would a *disease.* Rather, it is necessary to seek to *control* such a mechanism or break it down into its component parts, in the hope that we may prevent one or more of the units from entering into the appropriate convulsive "combinations" leading to a seizure.

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Our Clinic has been inclined to favor Foster Kennedy's idea of a "Spectrum of Epilepsy": The convulsive state may be so arranged that there exists a gradual shading of attacks from the *larval types* at the one extreme to the full-blown *major seizure* at the other. Some of these attack patterns are characteristic and easily recognized; others assume transition forms and combinations that often make them difficult of diagnosis and classification. (Table 1.)

The term *convulsive state* should include all types and forms of seizures, and, as long as an attempt is being made to get away from the word "epilepsy," this term seems to be a broad and satisfactory compromise. If escape from the ancient superstitions and fears surrounding the name of epilepsy alone were the object, it would seem better perhaps to adopt the simple, vulgar, but descriptive designation of a "fit," as it is frequently associated with other less fearful states such as "fits of laughter" and "fits of rage."

The most recent name suggested is the term *cerebral dysrhythmia.* Dysrhythmia refers to abnormal types of electrical brain waves with characteristic "spikes" and "frequencies" recorded by the electroencephalograph. The instrument is valuable in definitely differentiating between the four fundamental types of the convulsive state:

1. Larval state (psychic)
2. Petit mal
3. Jacksonian fits
4. Grand mal

As abnormal brain waves are also found in normal individuals during sleep, after hyperventilation, and as a result of disturbed sugar metabolism of the brain cells, it is obvious that nothing in the nature of pathognomonic evidence of epilepsy has been established. Where more than twenty individuals will manifest dysrhythmia, only one is afflicted with true convulsive attacks (and convulsive attacks occasionally arise or can be induced in normal individuals), so

TABLE I

THE CONVULSIVE STATE

(Symptomatic Progression)

Normal Integrated Level of Control

Unfavorable Procession of Symptoms ←

Favorable Improvement and Control of Symptoms →

Larval attacks (Psychic equivalent)	Aura; visual, auditory, olfactory, gustatory, vertiginous, epigastric
	Viscero-motor, pilomotor, vascular episodes
	Dreamy states, lapse, absence
	Psychomotor (sudden altered behavior)
	Recurrent amnesia and automatism
	Vacant spells; suspended mental activity
→	
Minor seizures (Petit mal)	Sudden transient *unconsciousness.* Micturition; objects dropped
	Temporary arrest of activity or movement
	Dilatation of pupils, pallor, staring
	Conjugate movement of eyes, turning head
	Sucking, champing, swallowing, vocalization
	Twitching face, arms, fingers, extremities
→	
Jacksonian attacks (Focal fits)	*Falling* (loss of "antigravity" muscle control)
	Full loss of consciousness (syncope)
	Rigid tonic phase, cyanosis, trismus
	Breath held (decerebrate rigidity)
	Clonic rhythmic movements of large flexor-extensor muscles
Major seizures (Grand mal)	Selective motor pattern unaltered by age or training
	Breathing re-established—sterterous type
	Cessation of movement—relaxation
→	
	Stupor (micturition, defecation, sweating, vomiting)
	Sleep followed by dull mental state
	Headache
	Mental deterioration

that it becomes obvious that further extended analysis, clarification, and research will have to be carried on before the designation of *cerebral dysrhythmia* can be reserved to the epileptic; or that the many others who show the "typical spikes" of dysrhythmia, but without seizures, can be proved to be "potentially" epileptic.

If the convulsive state may be considered analogous to a "spectrum of light" as Foster Kennedy pointed out, it is only of academic interest to name and classify the endless shades in the one, or the varieties of attacks possible in the other.

Diagnosis in the past has too frequently concerned itself with controversial borderline classifications, rather than attempting to establish a basis for therapeutic relief in the problem that confronts us today. There are, however, certain *common denominators* that underly the majority of seizures and should be sought for so that enlightened diagnostic and therapeutic considerations can be directed at the *source of trouble* rather than adopting blindly palliative sedation designed to conceal the progression of symptoms after they have finally developed.

Diagnosis in convulsions should concern itself not only with the distinguishing characteristics that separate the convulsive state from other conditions, but also with classification of its own types. Careful appraisal of certain *structural deviations* most commonly encountered in the physical make-up of those who suffer from so-called essential (idiopathic) seizures should be included in the field of survey. Thus the *diagnosis* of a *structural inadequacy* may offer a clue to certain contributory factors related to the seizures, whereas, designation of the condition as an "essential epilepsy" merely surrenders the problem into the realm of ignorance and despair.

Recently it has been realized that correction of certain common *structural inadequacies* of the chest and cardiovascular mechanism (through carefully planned exercise) has led to definite improvement in the patient and better control of the seizure, (more efficient cardiocerebral circulation, increase in carbon dioxide reserve).

DIFFERENTIAL DIAGNOSIS

True convulsive seizures are easily recognized even in their rudimentary patterns. Diagnosis chiefly concerns classification of types arising in the borderline groups of the *larval forms*. Here, sudden alteration of behavior, temper tantrums and violent emotional storms are occasionally believed to be "psychic equivalents" of a seizure and must be differentiated from purely psychopathic states. Careful observation of the patient is required to ascertain if the attacks are preceded or associated with *dilatation of the pupils, sudden short periods of loss of consciousness;* or followed by short episodes of *automatism*, brief *amnesia*, *micturition* and *confusion*. Certainty as to the diagnosis of larval attacks can only be established by careful tracings from the electro-encephalograph, or watchful waiting to determine if more definite and characteristic signs of transitory loss of consciousness and movements occur. (Progression of Symptoms, Table 1.)

Syncope itself is differentiated from *petit mal* by the prodromal symptoms of pallor, sweating nausea and weakness. The cause for syncope is usually apparent; inquiry reveals emotional disturbances, anemia, cardiac irregularity, hemorrhage or hypotension. The patient recovers more slowly from a syncopal attack. On the other hand, the loss of consciousness in *petit mal* is sudden and transient, the momentary pallor is replaced by flushing and the fixed stare and dilatation of the pupils promptly disappears. Recovery is almost immediate and usually the patient has no idea or recollection of the event.

Narcolepsy (Gelineau, 1880) is the term applied to an *imperious desire for sudden sleep* occurring at various intervals and of short duration. The attacks may occur at any moment, even during activities such as eating, walking, or boarding a street car. The patient usually wakes suddenly with a clear mind. *Bradycardia* has been noted

by Kovacs but pupils are not known to be abnormal. Ephedrine and benzedrine sulfate are helpful in treatment.

Cataplexy is characterized by sudden attacks of loss of muscle tone. The patient falls to the knees or ground *without* loss of consciousness. Attacks are known to be brought on by extreme emotions of fear, anger, laughing or weeping. The pupillary response to light is retained, but the corneal and patellar reflexes are said to be lost. Fortunately the condition is rare, as it is most difficult to control. Notkin and Jelliffe believe the condition related to epilepsy.

Pyknolepsy ("frequent" seizures) refers to sudden flutter of the lids, turning of the eyes, arrest in the stream of thought, but without falling or convulsive movements—authorities agree that so-called pyknolepsy is in reality only an early arrested phase of petit mal and no differential diagnosis is necessary.

Meniere's disease may give rise to acute attacks of vertigo and falling to the ground with transient unconsciousness. It is easily differentiated from *petit mal* by the history, tinnitus, nausea, profound state of collapse and progressive deafness that ensues.

Hysteria and *simulation* are easily detected as the patient *closes the eyes* firmly during the attack, pupils react to light stimuli and *do not dilate*. Movements of the arms and legs are bizarre and irregular. Cyanosis is absent or poorly substituted by periods of straining. Muttering, crying, slurred speech and prolongation of the dramatic attitudes simplify the diagnosis. Injury, biting the tongue, and micturition rarely occur in the malingerer or hysterical individual. The reflexes remain unaltered and postconvulsive headache and dullness are lacking.

DIAGNOSIS

Larval State. In this group should be placed those patients who may suddenly alter behavior so that the abruptness, the purposelessness, and the repetitive type of action strongly suggests something besides a "trend" toward a psychopathic state.

Where sudden periodic changes in behavior, personality, or activity arise out of an otherwise normal background, the present tendency is to consider such "temper tantrums," "behavior problems," or automatism as possibly related to the convulsive state—as based upon electroencephalographic evidence of dysrhythmia (Gibbs), but it must be recalled that similar findings are present in a wide variety of nonconvulsant types.

The "dreamy states" preceding or following well recognized minor or major convulsive episodes indicate, as Hughlings Jackson pointed out, that such manifestations may occur alone and in fact do, although the profession is more inclined to consider these in the realm of psychopathic disorders. The diagnostic boundary line in most instances is an artificial one and depends chiefly upon whether the symptoms are viewed by a psychiatrist or an organic neurologist. The use of the term "psychic epilepsy" or "psychic equivalent" seems to be the compromise of the moment.

Irritability, migrainous headache, outbursts of anger, personality changes, periodic jargon aphasia, amnesia and trance-like episodes are not necessarily confined to the early convulsive state. Many of these symptoms are common in organic lesions of the brain, as well as sequelae to severe head trauma, meningitis and encephalitis. They may follow a convulsive seizure, as well as precede it. Correction of some of these symptoms may be afforded by methods described below under *postparoxysmal state.*

Minor seizures (petit mal) are attacks characterized by sudden dilatation of the pupils, fluttering of the eyelids, deviation of the eyes, turning of the head, sucking or chewing movements of the mouth, swallowing movements, clucking and gurgling sounds with or without profuse salivation.

These minor seizures range from transitory moments of "mental lapse" or blurring of consciousness with an unnatural stare, to twitching and jerking movements beyond the patient's voluntary control.

Normal activities may be briefly arrested and then continue to original fulfillment of purpose without the victim being aware of interruption.

An arbitrary line may be drawn between a *minor* and *major* attack by the fact that the patient *does not fall.* Where sufficient loss of brain control permits the release of the "anti-gravity muscles," which maintain the postures of sitting or standing, it can be assumed that the attack is sufficiently profound to classify it in the major group, even though jerking movements are absent (cf. syncope and cataplexy).

Jacksonian attacks are *focal* fits characterized by sudden onset of localized *sensory aura* and, or, *motor responses, with* or *without* the subsequent *loss of consciousness.* Spreading of the sensory or motor phenomena in orderly progression from face, to arm, to leg, or vice versa, may be followed by a generalized major seizure. Twitchings or jerking movements may begin in the fingers, face, foot, et cetera before involving other parts. Usually some localized organic lesion of the brain is demonstrable by encephalography or operation.

Of most importance is the need for careful study in such cases to rule out cerebral neoplasm or other progressive organic lesion. In addition to the routine *neurological* examination, such a patient should have a *fundus* examination, *visual fields* charted, *x-ray* of the *head* with special films taken for *pineal* measurements (Chamberlain technic) in order to determine *displacement* of the gland if visualized. *Spinal puncture* with pressure reading, pneumo-encephalography (if no contraindication exists) along with routine blood, urine, and serological studies. The pneumo-encephalographic films will, in all probability, reveal the nature of the focal pathological process and determine whether surgical relief may be indicated.

Major seizures (grand mal) may be ushered in by a sudden cry, gurgling sound, or, without warning, the patient may fall unconscious striking the head and injuring other parts of the body. Stiffening of the muscles begins almost immediately, usually with extreme turning of the head. An *extensor rigidity* often manifests itself and closely resembles the "decerebrate" attacks associated with lesions of the peduncles. This is known as the "tonic" phase. The breath is held, the patient becomes livid or cyanotic, the *pupils* are *dilated* and *fixed,* the cornea insensitive, the *eyelids open,* teeth and fists clenched, the body often in opisthotonos.

In ten to thirty seconds quiverings and small rhythmical jerkings of the flexor muscles of the upper and lower extremities arise (the *clonic* phase of the attack). Nystagmoid movements of the eyes and contractures of the face begin with increasing violence, so that the body appears to be almost torn apart, the head beats upon the floor, the breath is now forced in and out by the abdominal muscles. The tongue, forced out between the teeth, may be caught in the clamping movements of the jaws and badly lacerated. Increased salivation, bleeding, and turmoil within the mouth often produces *bloody foam.* Sweating, vomiting, incontinence and stertorous breathing ensue and the attack usually subsides after one to three minutes into a *post-paroxysmal* state with accompanying stupor (profound sleep).

It is important to note that although major seizures may occur without one or more of the above symptoms, there is no deviation from the pattern of flexor-extensor type of muscle response whether observed in the infant or the highly trained adult. These movements suggest an early motor pattern seen in the swimming action of ambloblastoma. *Never* do rotary, prehensile skilled movements, such as we see in chorea or athetosis, occur in a true convulsion. Differential diagnosis between

malingering, simulation and hysteria can be made upon this characteristic motor pattern alone. Dilated pupils, open eyelids and incontinence at the proper moment cannot be successfully imitated.

When *major convulsive seizures* are associated with *organic alterations* in the neurological findings—brain tumor, abscess, meningitis, trauma, edema, subarachnoid hemorrhage and local irritation to the brain surface must be considered and ruled out before the *diagnosis* of *grand mal* becomes valid.

Postparoxysmal State. The stupor and relaxation which follows a major seizure has been considered an "exhaustion phenomenon." Here again, closer analysis would indicate that appearances sometimes deceive use. When "status epilepticus" occurs, seizures may go on steadily for hours without signs of cortical exhaustion. Belief that the attack should be terminated in a few minutes because of inability of the motor mechanism to continue is far from the case in strychnine convulsions (spinal release), whereas those seizures produced by repeated "electric shock," profoundly exhaust the brain, and a period of recovery is required before another attack ensues. Subsequent convulsions can be induced repeatedly with appropriate stimuli in most instances of convulsive seizures, in which secondary changes in cerebral circulation and edema are not present.

The attack terminates in a state of profound sleep or stupor due to one of two fundamental reasons: (1) Sufficient circulation returns to the higher cortical levels to *re-establish* adequate metabolism and functions of *control* over the more primitive motor patterns, or (2) edema, pressure, and anoxia build up in the brain tissues to such a degree that circulation to the cortex is insufficient to maintain further metabolic function to the motor elements involved and movements cease with signs of temporary flaccid paralysis as in a "stroke." It may be true that the motor cells "exhaust" their available supplies of oxygen and energy, but from a prognostic standpoint, the important feature to bear in mind is that *all* of the cortex and *other* ganglion cells devoted to *memory, hearing, integration* and *higher intellectual functions* are sharing in this generalized widespread edema, pressure and anoxia, so that instead of the concept of simply motor elements alone becoming exhausted, such as seen after violent exercise, the brain as a whole suffers severe damage, and *progressive mental deterioration* develops relentlessly with recurrent seizures.

The postparoxysmal state must, therefore, be carefully observed by trained personnel or someone in the family delegated to record duration and events. It is the period of stupor and anoxia that is disastrous to the brain, not the fit. If this phase can be shortened or terminated by general measures of dehydration, the patient's prognosis from the standpoint of mental integrity is greatly enhanced.

It will be found that at one extreme of this postparoxysmal phase we have *somnambulistic* behaviors, in which walking, running, dressing and undressing, fighting, automatism and repetitive movements and acts indicate an altered coordination of consciousness, but still sufficient retention of cerebral circulation and metabolism to carry on most of the fundamental processes of *upright posture*, balance, speech, movement and even good judgment such as demonstrated in avoiding obstacles and solving problems involving passageways, stairs, shoe laces, buttons, et cetera.

On the other hand, patients manifesting incontinence of bowels or bladder, wide pupils, divergences of the eyes, loss of deep tendon reflexes, positive Babinski sign, or stertorous breathing and pulse pressure above 50 mm. of mercury are not unlike individuals suffering from the severe effects of *concussion* due to head trauma. Such patients should be treated for *cerebral edema* along the lines established for head trauma (immediate and complete spinal drainage, strict fluid limitation, appropriate tissue dehydration (purgation and

sweating), regulation of salt, sweets and diet) if progressive mental impairment is to be avoided.

Fig. 1. Pneumo-encephalogram in the sitting position showing air in the subdural space, which has replaced an abnormal collection of cerebrospinal fluid. Note displacement of the brain away from the sinus and the consequent stretching and narrowing of the attached cortical veins. Fluid level can be seen gravitating toward the base. In this instance, air introduced by the spinal route has found access to the subdural space through some tear in the arachnoid. This type of subdural hygroma is, therefore, drainable by spinal puncture. Other supracortical collections must be drained directly through trephine opening of the skull.

As appropriate treatment depends upon proper diagnosis, it is obvious that sedation and temporary supervisional restraint (often increase in sweets and forced feedings) are needed to "slow down" the "psychic equivalent" type, whereas diametrically opposed therapy of *activity*, *elimination*, and restrictions of food and fluid are required for the "stupid" stuporous type.

There are patients that manifest prolonged periods of postparoxysmal *hallucinations*, *amnesia*, *excitement*, *incoherence* and *violent impulsive acts*. These manifestations are not unlike those encountered in *delirium tremens* and are probably due to the same cause; namely, abnormal collections of clear subdural fluid (hygroma). If bilateral trephine is done in the temporo-occipital area with the patient in the sitting position (fluid gravitates to the base), large quantities of fluid can be drained off from over the arachnoid and cortex.

Spinal (subarachnoid) fluid escapes into the subdural space from a "leak" in the arachnoid, probably forced out during the violent stage of the seizure. This fluid is not drainable by spinal or cisternal puncture as it lies in the *subdural* space. It tends to gravitate to the temporo-occipital areas (visual-auditory zones) as the patient usually lies on the back (restrained) but the fluid may be moved to the frontal poles if the patient is placed face downward.

Ounce for ounce, the disturbance in blood volume relationships by this abnormal collection of fluid within the skull is the same as that of a clot, tumor or abscess. The only difference is that spinal fluid is clear and mobile giving rise to fairly generalized symptoms of disturbance, whereas a clot is blue-red and circumscribed, giving rise to focal signs in advance of the generalized circulatory impairment.

At necropsy the subdural clot is easily demonstrated, whereas the fluid trickles away while the skull and dura are being removed, leaving the pathologist nothing "significant" to report and the mystery unsolved. Since operating on such types in the sitting position (fluid gravitates to the base), draining the subdural space for thirty-six hours, the symptoms of hallucinations, excitement, amnesia, and mental torpor promptly disappear and are correctable in most instances as long as the subdural fluid collections can be prevented from recurrence (curtailment of liquid intake, solid or dry diet).

With encephalographic demonstration of the presence of abnormal collections of subdural fluid in a large number of these types, the diagnosis has been confirmed by neurosurgical verification, and sympto-

matic improvement follows appropriate methods of drainage. (Fig. 1.)

Thus a large number of postparoxysmal manifestations fall chiefly into three types of altered cerebral circulation: (a) Prompt and almost adequate return of circulation (metabolism). The patient may recover consciousness promptly with clear mentality or only transitory confusion; (b) impaired cortical capillary network circulation (subdural fluid, "wet brain") and venous stasis (narrowing of veins due to displacement of the hemisphere away from the attachment of the veins entering the dural sinuses). The patient may manifest prolonged abnormal sensory or motor manifestations before or after attacks (c) Intrinsic swelling and edema of brain tissue due to profound anoxia and pressure. The patient appears to be severely "knocked out" by the seizure but recovers gradually. Headache, vomiting, dullness and mental deterioration are commonly associated with this group.

Various degrees and combinations of the above altered physiological relationships have been observed at operation in psychotic states with or without convulsions. The "psychic equivalent" group no longer rests upon "unknown pathology" but requires a diagnostic pneumo-encephalogram and volume displacement measurements to establish the diagnosis, as well as correct the underlying circulatory disturbance.

CIRCULATORY CONSIDERATIONS PREDISPOSING TO SEIZURES

Sharp exsanguination, sudden clamping of the jugular veins, or compression of the arteries in the neck may produce convulsive seizures (Kussmaul and Tenner*). Failure of the ventricle of the heart to beat for three to seven seconds (pulse 8 to 20 per minute) may produce unconsciousness, and *asystole* lasting for fifteen to twenty seconds is frequently associated with convulsive seizures (Sir Thomas Lewis*).

Thus careful examination and observation must be carried out to detect *cardiac syncope* as well as cerebral circulatory insufficiencies that may be intermittent or chronic. The following considerations must enter into every obscure problem of convulsive seizures presented for diagnosis:

Cardiocirculatory Insufficiencies:†

I. Cardiac Syncope: (Intrinsic disturbance of output of the heart in spite of adequate venous return)

(a) *Stokes-Adams Syndrome* (arteriosclerotic background)

(1) Severe paroxysmal bradycardia

(2) Ventricular standstill

(3) Vagal attack plus ventricular fibrillation

Carotid sinus reflex

Habitual slow pulse

Extrasystoles

(b) Partial *heart block:* Severe lapse of ventricular beat (intermittent and recurrent asystoles produce fits at variable intervals; prolonged episodes associated with a combination of (a) and (c) may produce "Status Epilepticus"). The convulsions usually occur without warning (aura), incontinence and tongue biting are rare, bradycardia is present at the time of the attack and the transmitted auricular contractions may even be visible in the veins of the neck.

(c) *Tachycardia or Ventricular Fibrillation:* When the rate reaches 200 or more per minute, little or no blood is forced into the arteries, blood pressure falls and cerebral circulation fails. *Spasm* or occlu-

* Kussmaul and Tenner. The Nature and Origin of Epileptiform Convulsions Caused by Profuse Bleeding. Trans. E. Bonner. London, 1859. New Sydenham Soc.

* Lewis, Sir Thomas. Osler's Modern Medicine. McCrae IV, 3d ed. 402. Philadelphia, 1927. Lea & Febiger.

† Roesler, Hugo. The author is indebted to Dr. Roesler for suggestions and assistance in the arrangement of the cardiocirculatory insufficiencies.

sion of the *coronary artery, wounds of the heart, poisons, electrical shocks* and large doses of suprarenal extract may precipitate fibrillation.

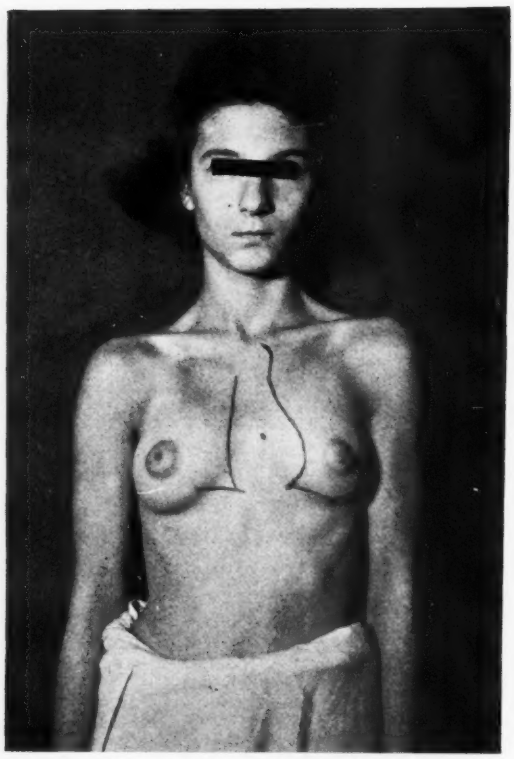


Fig. 2. Structural inadequacy showing long neck, small chest and heart; ten-year old pattern, with development of extremities and adult relationships elsewhere. (A. O., age fourteen.)

(d) *Aortic Stenosis:*
  (1) Congenital
  (2) Acquired—trauma, aneurysm, sclerosis

II. Relative Cardiac Syncope: (Inadequate capacity of heart in spite of normal mechanism)
  (a) Small infantile hearts (as shown by orthodiagrams). Small underdeveloped carotid arteries
  (b) Disproportionately long neck, increase in distance between the aortic valve and vertex of the skull, demanding greater than normal cardiac effort or capacity. *Circulatory stasis* and insufficiency arises *at the brain level* if blood pressure falls even moderately in this group. (Fig. 2.) It is obvious that a small heart, failing to deliver blood to the brain because of a greater height and distance to overcome than in the normal, presents the same potential type of circulatory stasis as far as the brain organ is concerned as the mechanism of asystole would to an otherwise normal heart and vascular set-up (cf. Stokes-Adams syndrome).
  (c) Stenosis of carotid canals in the skull, compression of both the carotid and vertebral arteries by tumors, cellulitis or swelling. Vascular anomalies, aneurysms and abnormal patterns which profoundly impair total cerebral circulation.
  (d) Relative structural inadequacies of chest and heart due to rapid growth of head and extremities in puberty or adolescence. (Six to seven year old chest and heart size with arms, legs, neck and head of fourteen to sixteen year-old pattern)

III. Vascular Syncope: (Insufficient return of venous blood for required output and pressure)
  (a) *Exsanguination and profound anemia*
  (b) *Splanchnic dilatation and shock*
  (c) Compression or obstruction to the *vena cava*
  (d) Adrenal insufficiency
    (1) Acute hemorrhage
    (2) Chronic (Addison's) disease

IV. Jugular Occlusion: (Impairment of cerebral venous drainage, stasis and edema at the brain level)
  (a) *Acute thymic swelling* with compression of both jugulars passing through the bony thoracic ring

(b) *Swellings* in the neck (metastatic tumor, paratonsillar abscess, cellulitis)

(c) *Traumatic* or *infectious thrombosis* (where stenosis of the jugular foramen or underdevelopment of one jugular outlet (as shown by *x*-ray) exists, occlusion of the "dominant" sinus* or vein may result in cerebral edema and "Status Epilepticus."

INTRACRANIAL FLUID CONSIDERATIONS PREDISPOSING TO SEIZURES

A. "Toxic" Types: (Water intoxication). Convulsions that arise during an acute illness; or for some so-called "toxic" cause, will be found in most instances to be associated with a profound disturbance of water metabolism and coincident "wet" brain. When renal elimination becomes impaired, or the escape of moisture from the skin surface is curtailed (as in fever or cold weather), a state of water retention arises similar to that induced experimentally by Fremont-Smith, McQuarrie and others. They have demonstrated that convulsions in animals and human beings may easily be induced by forcing fluids (mouth, vein, or subcutaneously) after temporarily shutting the kidneys down with injections of posterior lobe pituitary extract. This is essentially the same mechanism as that spontaneously encountered in eclampsia, in which pituitary dysfunction, hypertension and retention of fluid associated with faulty renal and skin elimination give rise to convulsive attacks (Fay, Arnold,† Behney). If other vasospastic or arteriosclerotic processes are deemed responsible for the renal failure, the term "*uremic*,"‡ or "*toxic*" or "essential" is usually applied to the convulsions.

It is now believed that the convulsion is not produced by "poisons" or vague "toxins" but rather by a *disturbance of electrical cellular potentials, oxygen, permeability* and *conduction*. The rising tide of intracranial fluid permits displacement of circulatory blood volume and consequently metabolic alterations, failure of controlling areas through circulatory stasis and finally, release of primitive motor patterns as the externalized manifestation of a seizure.

B. Hydration Types: (Fat, water-logged, hypogonadal types). A state of *water intoxication* (Rowntree) may be easily induced in certain persons and animals by the simple process of *overindulgence in fluids* when the portals of elimination are inadequate. Typical convulsive seizures arise and may even progress to a fatal form of "*status epilepticus*" unless satisfactory drainage of fluid from the subarachnoid spaces (spinal puncture), the subdural space (trephine), and the interstitial brain spaces (dehydration) can be promptly accomplished.

In this type we find the "wet brain" of *trauma* as well as the *alcoholic* "wet brain" (in which mild trauma precipitates edema of brain tissue on top of overfilled subarachnoid channels; or ruptures of the arachnoid permitting the formation of a subdural hygroma). "Wet brain" may also be associated with severe venous stasis, due to occlusion or inflammation of the dural sinuses (superior and lateral, or cavernous), *constriction of the jugular veins* in the neck or chest, by *thymic enlargement*, or *thrombosis* from mastoid disease.

Certain *drugs, allergies* and *anesthetics* may produce a rapid increase in intra-

* In 85 per cent the right lateral sinus and jugular are larger than the left. In approximately 5 per cent one jugular is so large and "dominant" that if occluded, circulation cannot be taken over sufficiently by the smaller remaining vessel and delay and stasis in the brain tissues may result.

† Arnold, J. and Fay, Temple. Eclampsia; its prevention and control by means of fluid limitation and dehydration. *Surg., Gynec. & Obst.*, 55: 129–150, 1932.

‡ I have observed patients with high blood urea (in one instance 110 mg. per 100 cc.) without loss of consciousness or convulsive seizures and, on the other hand, have seen convulsions most frequently when the blood urea was only slightly elevated, if at all. In either instance, in which convulsions exist, trephine or encephalogram has revealed increase in the volume of subarachnoid; or subdural fluid (wet brain), with or without increase in spinal pressure. The operative findings are identical with those of "water intoxication."

cranial fluid due to transudation into the tissue spaces.

It is striking with what rapidity *ether* brings about increase in subarachnoid (spinal) fluid and actual swelling of the brain.*

Although some vague "toxic" agent is usually said to be the cause of the convulsive seizures, one of the outstanding common denominators so far evident in widely separated clinical states, concerns intracranial changes in intracranial fluid *volume* and cell *permeability* (altered cell surface potentials and electrolytes—Spiegel and Spiegel-Adolf) similar to those noted in *water intoxication* itself.

It is evident that the water *per se* is not responsible for the seizure. The attack arises from *altered physiology of tissue, conduction* and "*threshold of control,*" which are all affected by lack of oxygen, altered sugar and carbon dioxide metabolism and finally to inadequate intracranial blood volume and circulation due to displacement by the rising tide of fluid.

As the walls of the craniovertebral cavity cannot expand, an increase in fluid volume in the tissues of the brain or subarachnoid spaces can only mean an equal displacement of some other volume (Monro-Kellie). At the moment, at least, this chiefly concerns blood volume.

As the fluid volume rises within the skull (obvious in the open fontanel or area of decompression of the skull), circulatory blood volume is diminished so that the loss of higher controlling centers may allow the patient even to become unconscious.

It is known that drugs or physical agents which tend to increase cellular permeability to fluid *favor convulsive seizures* in the predisposed case, whereas drugs which tend to prevent easy permeability of fluid into the cells *prevent attacks* (Spiegel, measurements of the delta). When the inhibitory and restraining influences of the higher cortical levels are diminished, normal or abnormal stimuli may reach and discharge the motor patterns otherwise held in constant control.

An experience familiar to all will serve to illustrate a familiar normal mechanism: If a gun is fired close behind a person unaware of such a possibility, a sudden generalized "defense response," such as jumping, screaming, et cetera, usually ensues. If, however, the individual is shown the gun and has time to set and "control" the motor mechanism, no overt response occurs. Thus the same stimulus that reached the brain at an unguarded moment and produced a startled spasm, is harmless in its effect when anticipated and properly controlled. If repeated "jumping" movements were to occur suddenly without a detectable noise or cause, the individual manifesting such actions would undoubtedly be suspected of having "epileptoid" tendencies. The electro-encephalogram has shown bursts of abnormal waves may surge through the brain without external stimuli and the motor discharges which follow are thought to be due to these spontaneous waves.

CONVULSIONS IN INFANTS AND CHILDREN

Almost two-thirds of all convulsions occur within the first two years of life. Comparatively few seizures arise between the years of three to seven. After this time, disproportionate growth (inadequacy of circulatory function) and puberty predispose the child to convulsions. Only about 23 per cent of attacks arise after the completion of the growth period (eighteen to twenty years female, nineteen to twenty-two years male) and these are usually easily identified with demonstrable organic lesions (so-called *symptomatic epilepsy*).

* With the human cortex exposed, (the patient under avertin, local anesthesia, quiet and relaxed on the operating table) we have often observed swelling of the brain begin within thirty seconds after a few whiffs of ether had been inhaled. As this increase in tissue fluid volume occurs in the absence of straining or holding of the breath, and will continue if not checked to actual protrusion of the brain above the scalp level, it indicates the degree of danger from intracranial pressure that exists when *ether* is given to patients in whom an hydration state already exists, also the rapidity of fluid accumulation possible under certain circumstances.

Diagnosis is usually most difficult in the infant group because of the many limitations for adequate study due to size, age and lack of co-operation.

The so-called "cause" for infantile convulsions can usually be found associated with one or more of the following significant *predisposing factors:*

I. Birth Trauma:

(a) *Asphyxia of brain tissue* (lack of oxygen for ninety seconds or longer)

1. Delay in breathing after delivery
2. Imperfect circulation, cardiac anomaly or defect (blue baby)
3. Obstruction of jugular return from brain
   (x) Delay in rotation of after coming part (compression or twisting of neck)
   (y) Strangulation by cord or forceps
   (z) Structural anomalies, thymic enlargement

(b) *Subarachnoid hemorrhage* (bloody spinal fluid)

1. Increase of pressure due to retention of spinal fluid
   (x) Hydration
2. Communicating hydrocephalus
   (x) Generalized at vertex or base, mental retardation, delay in walking and talking
   (y) Focal
      Unilateral (hemiplegia—flaccid or spastic)
      Bilateral (spastic diplegia—Little's disease)
3. Porencephaly
   (x) Intracerebral hemorrhage or thrombosis with liquefaction of cerebral tissues and eventual rupture into the ventricle or to the surface (up to third month of life), occasional cortical cyst and scar formation

(c) *Subdural collections*

A. *Hemorrhage* (clear spinal fluid)

(1) Rupture of tentorial veins, clot at the base (vomiting and generalized symptoms; stiff neck and stupor)

(2) Rupture of cortical veins, clot over hemisphere (irritability, dilated pupil on side of clot, focal signs with paralysis, and stupor)

(3) Rupture of arachnoid, combination of blood and fluid covering one or both hemispheres

B. *Hygroma—"wet brain"*

(1) Spinal fluid escape into the subdural space. (Myotic pupils, dullness, prolonged attacks with tendency to "status epilepticus" followed by generalized flaccidity and stupor). Such collections of fluid are not drainable from the spinal canal (unless wide rupture of the arachnoid at the base is present). Direct approach through the skull is required to save the patient's life.

II. Cerebral Venous Stasis:

(a) Enlarged thymus (jugular compression at the first thoracic ring)
   (x) Status lymphaticus (often fatal)
   (y) Laryngismus stridulus (diphtheritic)

(b) Swellings of the neck
   (x) Infections
   (y) Edema
   (z) Hematoma

(c) Congenital absence or stenosis of the jugular foramina of the skull.

(x) Nocturnal attacks when sleeping on abdomen with neck sharply turned

III. Circulatory Inadequacy (Cyanosis, murmurs, enlarged heart):

(a) Patulous foramen ovale

(b) Aortic stenosis

Clinical findings, alteration in heart size, murmurs, etc., make diagnosis possible

IV. Acute Brain Infections:

(A) *Direct Involvement* (Inflammation, edema, intrinsic irritative and destructive lesions, local and generalized seizures; outcome depends upon the disease)

(a) *Encephalitis* (fever, headache, dullness *or* irritability, twitching of muscles, double vision, loss of convergence, oily skin, atonic facies)

(b) *Meningitis* (fever, stiffness of the neck, headache, vomiting, increased spinal pressure; choked disc, convulsions are usually generalized)

(1) Suppurative

(2) Tuberculous

(3) Cerebrospinal epidemic

(4) Virus—hydrophobic tetanus

(B) *Indirect Involvement* (Paradural reaction, effusion, wet brain, edema, water intoxication type)

(c) *Mastoiditis and Sinusitis:* Usual history of running ear or upper respiratory infection, swelling of anterior and posterior cervical glands, fever, headache, vomiting, slow pulse, marked tenderness over carotid arteries in the neck, convulsions are Jacksonian or generalized, trephine discloses marked "subdural wet brain"

(d) *Whooping Cough:* Venous back pressure produced by prolonged spasms of coughing as well as cyanosis and asphyxia. Subarachnoid hemorrhage may arise due to excessive straining precipitating focal seizures, "status" or paralysis

(e) *Scarlet fever, diphtheria, measles and other acute toxic states:* Convulsions may be serious and even fatal unless the acute "wet brain" characteristics underlying the attacks are recognized. Toxic renal shut-down with no skin elimination (dry, feverish) favors water intoxication and "wet brain" especially if fluids are forced in this period. (Treatment: purgation, sweating, fluid restriction, *spinal drainage* or subdural drainage)

V. Chronic Brain Infections: (Subarachnoid fibrosis, communicating hydrocephalus, and wet brain type)

(A) Congenital syphilis—(Wassermann may be negative); structural and intrinsic lesions, scars and atrophy, stigmas of degeneration

(B) Tuberculosis—arachnoidal scars, isolated tubercles and zones of inflammation (granulomas)

(C) Postmeningitic adhesive arachnoiditis

Symptoms and diagnosis similar to post-traumatic group. Diagnosis established by encephalogram along with history, clinical and laboratory findings.

VI. Metabolic Disturbances:

Calcium Deficiency (Blood Calcium below 8)

(a) Parathyroid insufficiency
(b) Rickets
(c) Spasmophilia
(d) Tetany

Sugar:

(a) Hyperinsulinism (excessive therapy)
(b) Adenoma of the pancreas

Carbon Dioxide:

(a) Hyperventilation (blowing off of carbon dioxide)
(b) Alkalosis (lack of acid or carbonic acid).
(x) Excessive sodium bicarbonate, etc.

Water:

(a) Hydration states
(b) Hypotonic solutions (intravenously)

VII. Drugs, Poisons (From mother or environment):

(a) Lead encephalopathy (eating paint off cribs, occupational, accidental)
(b) Strychnine
(c) Camphor, picrotoxin, metrazol, alcohol, thujone, absinthe
(d) Electricity
(e) Insulin, pitressin

Many of the above considerations apply equally well to the adolescent or adult. It must be emphasized that the higher incidence of attacks in infants and children is probably due to the immature state of development of the *controlling centers* in the brain. Sensory stimuli are more intense (unmodified or dampened) and there is less control over the "defense" and subcortical reflex motor patterns.

Thus some of the older concepts may be understood if given one or more of the above predisposing factors, the aggravation caused by "teething," worms, phimosis, irritating foods, etc., could easily upset the unstable cerebral controls of early life. During this period, sudden loud noises may produce violent responses that would be entirely controlled in later life. It is well known that in strychnine poisoning the sound caused by the slam of a door, or clap of the hands may be enough to precipitate a convulsive seizure. Thus a common impulse (sound, light, pain, vibration, touch) may be all the stimulus necessary to "touch off" an attack if the control mechanism is withdrawn. "Thresholds" for control of motor patterns are far from developed at birth. In the legs these are not complete until walking is established.

COMMON DENOMINATORS CONCERNED WITH CONVULSIVE SEIZURES

Summarizing some of the inter-relationships of oxygen, water, and carbon dioxide in the predisposing of an individual or animal to an attack, the chart on page 328 (modified from Lennox and Cobb) is most illuminating.

Anoxia favors increased permeability of the capillaries to fluid, giving rise to rapid transudation of fluid into the tissues with consequent swelling. This increased fluid volume pushes out still more blood from the cranial cavity causing more anemia of the brain and a vicious cycle with mounting anoxia and further edema.

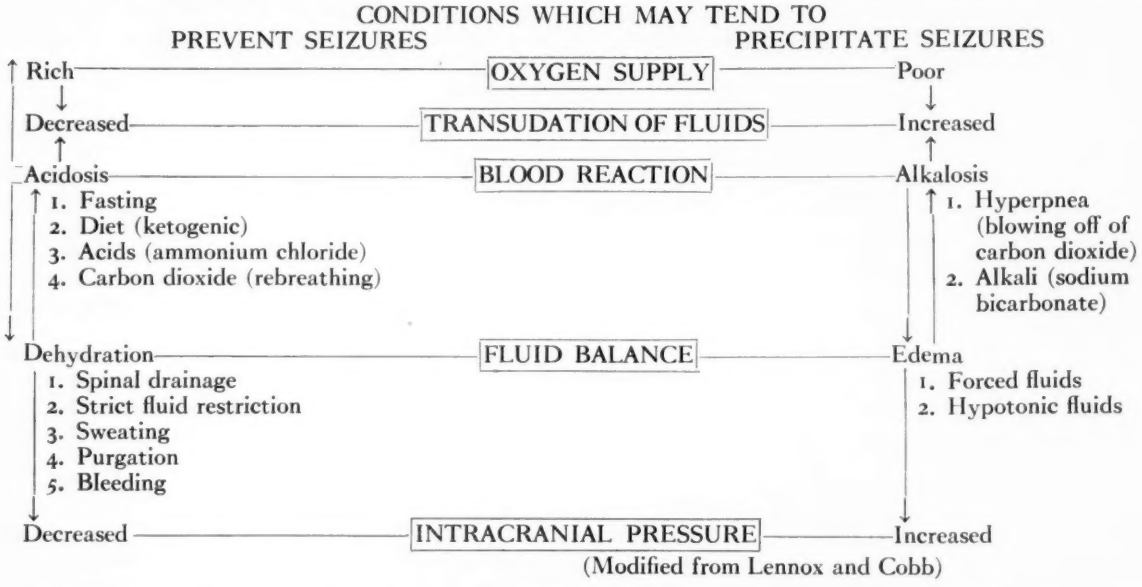
Now that everyone can enjoy a convulsion when we choose to produce it, the obvious fact that a perfectly normal and adequate brain may suffer profoundly from purely extracranial causes (viz., "poor plumbing" system for blood delivery and drainage) or inadequate nutrition and circulation, might well justify the organ in "having a fit" over the fact that its existence and survival is constantly jeopardized.

We have studied and observed more than 1,200 problems of the convulsive state in all of its clinical neurological and neurosurgical aspects during the past sixteen years. Aside from the organic cases, 820 so-called epileptics were treated along the lines suggested by the diagnostic disclosures indicated above. Our emphasis has been on correction if possible of the physiological, structural, chemical or metabolic disturbance. Three out of four of

these patients have found satisfactory improvement and control.

The objective has been to apply to the brain organ as a whole the first principle of medicine or surgery, "An organ functions and heals best when it has a normal and adequate blood supply."

Medication should be restricted to a minimum (phenobarbital ¼ to ½ gr. two or three times daily). We believe that larger doses "slow down" the patient as well as obliterate the seizures. It has seemed to us it is more important to observe the favorable recession of seizures



(Modified from Lennox and Cobb)

Where small hearts existed, the patients have been put on the rowing machine, the track, routine heavy exercise. Deep breathing and "pull-ups" have helped to develop the chest.* Where hydration states exist (overweight, "one pint equals one pound"), appropriate curtailment of liquids and correction of diet has helped to prevent chronic "high tides" of fluid in the brain cavity. Toxic states with "water intoxication" patterns require temporary curtailment of liquids by mouth, vein or bowel, or drainage of spinal fluid as well as improvement in the ultimate portals of elimination (skin and bowels).

* The author is indebted to the Y. M. C. A. for their intelligent assistance in the correction of many relative inadequacies of the growth period. One must expect a short interval of increase in number and severity of attacks if sudden transition from a state of quiet sedation to one of strenuous exercise is attempted. Attacks due to "hyperventilation" can be overcome by teaching deep breathing and by increasing the size of the chest in order to establish an adequate carbon dioxide reserve, so that the ordinary demands of excitement and activity can be met without danger of a seizure.

in the order of improvement and *control* (Table 1) than it is to expect the drug to "cure" the condition by removing the seizure from before our eyes. As the convulsion is a *symptom complex*, drugs are only of temporary value and cannot be considered "curative" as quinine is for malaria.

As in other clinical problems (diabetes, syphilis, etc.), signs and symptoms may improve before they disappear. Those that consider any and all attacks of equal importance and fail to press the direction of treatment as improvement indicates will find that the use of drugs only permits the conditions underlying the attack to become worse, so that more sedation eventually is required and a false sense of security permitted to arise.

Too many members of the profession today indulge in the practice of "wishful thinking" in hopes that the condition will eventually correct itself or that the patient will "outgrow" or "outlast" the "disease." Such spontaneous physical adjustments

are known to occur and should increase our desire to hasten their fulfillment.

In considering *diagnosis in convulsions*, what formerly was a simple matter of differential diagnosis, and an individual matter as to expediency in selecting a name ("epileptic" or "epileptoid," if the supposed cause was easy to find; if obscure, "cryptogenic," if unknown "idiopathic") has now required the most important far-reaching and exhaustive analysis and attack upon the problem.

Success or failure in treatment of the patient may depend upon full and proper diagnosis. The former "causes" now become "predisposing factors." No longer do we speak of brain tumor or cortical scars as the "cause" of the seizure. It is true that their presence has altered local physiology, but removal of the tumor or the scar *does not always bring relief from the attacks*. Convulsions occur most frequently without the assistance of tumors or scars. The old "Post hoc propter hoc" weakness in medical reasoning still deludes those who allow their visual impressions to stop their continued contemplation of the problem.

It is, of course, evident that the scar or tumor is present every minute of the day and night and yet the attacks may be far apart or only once a week. If the lesion itself were the cause of the attacks, its *constant* stimulus should produce *constant* and continuous convulsions. To explain away this fact the idea of a "Lyden jar or battery" has been advanced by those who claim that energy is stored up until the discharge level is reached. "After discharge it takes time to again charge the battery." Anyone who has watched a case of "status epilepticus" has hoped and prayed that the "battery" would rapidly become discharged and the convulsions cease. Unfortunately, the explanation is not sufficient, for it is as unreasonable to expect to exhaust a convulsive mechanism (unless circulation to the motor areas is impaired) as it would be to expect a runner or swimmer to stop after a few minutes and wait days in order to proceed with muscular activities again.

In all of the organic processes we know associated with convulsive seizures, there has always been the additional unknown "X," the strange factor that came and went without a trace. As we find more and more "normal" elements in convulsive manifestations (which we have too long considered pathologic), we shall begin to speak of "normal" convulsive seizures when viewed dispassionately and compare these "standard types" with those of "atypical" character.

The prejudice with which the profession has viewed the mechanism of a convulsion as an "*evil*" and *undesirable affliction* of man is both unreasonable and unsound. The mechanism of labor is an unpleasant thing to watch and yet science has established its normal and abnormal characteristics. The possibility of this mechanism is inherent in every female but latent until called into active need. The same may be said of the convulsion, as it is inherent in both human and animal life and perhaps at one time in the long evolutionary process it, too, may have served a useful purpose.

At the fish level, head, eyes, tail and paddle movements join in convulsive efforts ("like a fish out of water") to regain the protection of the seawater with its abundance of electrolytes and oxygen—at the human level, to regain for the brain organ this same intrinsic need.

The "work-out" that a major convulsion gives to a patient with the coincident sweating, micturition and defecation rivals some of the best health resorts and elimination fads of the day. If it were not for the unconsciousness and "purposeless" movements (Piscian-Amphibian movements), one might compare the effort and violence at times with the running of the Olympian 100 yard dash.

Convinced that the profound mechanism of a seizure has not been developed or retained in the nervous mechanism throughout the evolutionary ages without some beneficial reason or purpose (cf.

the "evil" vermiform appendix as a vestigial organ, or the complicated actions seen in the vomiting mechanisms and those of shivering, fever and others, which have all eventually been explained and accepted as "normal" although at one time regarded as pernicious), it seems reasonable, in certain inadequate types, that we might anticipate nature's need for better development and function by *forcing exercises* in order to build a better circulation (athletic "Work-outs" instead of medicinal "black-outs"). Either because of, or in spite of, this new approach to the problem, we are now controlling 16 per cent more of the former "hopeless hereditary types" of the convulsive state than we did ten years ago and certainly what we have done, others can also accomplish, provided they approach the problem with an open mind and a diagnostic determination.

The same sound clinical view should prevail in dealing with the convulsive state as that which is maintained in diabetes; namely, that occasional reappearance of sugar in the urine or a momentary return of high blood sugar is not necessarily evidence of failure of the method of treatment, rather it is an indication to pursue with more care and detail the program responsible for bringing the more serious aspects and symptoms under definite control.

Those patients or physicians who merely count the *number* of attacks, as a basis of measuring the value and possibilities of treatment, often become discouraged during the most important and critical phase of improvement, when many minor attacks may take the place of a single major seizure (ratio about 3 to 1).

Minor seizures have not, in our experience, been associated with mental deterioration, whereas, the major seizure, with its subsequent period of stupor, has frequently been associated with bodily injury and serious regression of mental processes.

In our experience* the severity of the case is not to be measured by the number of attacks or the violence of the muscular response, but rather by the length of the period of stupor and sleep, following a seizure. As the patient improves, this period of unconsciousness shortens until finally attacks without loss of consciousness may occur or minor seizures without falling intervene. Conversely, if the attacks are associated with increasing periods of stupor and sleep, the patient's condition may be considered as progressing unfavorably.

Careful observations regarding the duration of the true period of unconsciousness should be recorded and compared from time to time, in order to evaluate the efficacy of treatment properly. It is, therefore, desirable to observe from time to time periods free from the use of drugs to determine what progress may have been made by corrective physical measures.

DISCUSSION

We have become convinced for many reasons that a convulsive seizure is closely associated with a selective degree of stasis or failure in cerebral circulation.

For the purpose of analysis and treatment, we have considered the seizure as a "protest" on the part of the nervous mechanism against circulatory failure as a serious threat to its integrity. Thus, after correction of inadequacies and improving circulation and routine, a large group of individuals, formerly considered as suffering from "essential," "idiopathic," "cryptogenic" or with other obscure so-called "hereditary" backgrounds, have in reality revealed themselves to be transient problems of physiohydrodynamics based upon structural inadequacies and ineffective levels of circulatory volumes within the cranial cavity.

We continue to emphasize that if a full diagnosis is to be made and therapeutic agents intelligently planned, a relentless

* From personal observations on 823 patients with symptoms of convulsive attacks hospitalized for diagnostic study, and based on 1,640 encephalographic and volume replacement measurements in the organic and convulsive groups.

search must be undertaken for those factors which might impair cerebral circulation and thus "*predispose* the individual *toward* a *convulsive seizure.*"

Twenty-two per cent of 2,000 patients studied by Lennox had important contributory causes as follows (symptomatic epilepsy):

	Per Cent
Congenital brain defect or birth trauma	5.6
Brain injury after birth	5.7
Infection of the brain	4.2
Tumors of the brain	2.6
Defect in brain circulation	1.9
Miscellaneous brain lesions	1.5
Conditions outside the brain	0.9
	22.4

Our clinical studies have revealed that 84 per cent of patients revealed important contributory causes if the additional factors surrounding disturbed water balance, circulatory and structural inadequacy, volume displacement and pneumoencephalographic alterations from the normal are also taken into consideration.

The clinician must include in his survey of "conditions outside the brain" the circulation from the heart, through the structures and vessels of the neck into the skull; within and around the brain, back along the venous pathways to the chest and heart again.

We have only to call attention to the Stokes-Adams syndrome or severe bradycardia; to the carotid-sinus-symptom-complex of altered arterial pressure and flow; to compression of the jugular veins by an enlarged thymus at the level of the bony ring of the first rib; to postpharyngeal abscesses, swellings and occlusions in the neck; to congenital absence and anomalies of the jugular foramina of the skull, as well as to the many direct and indirect causes for alteration of circulation within the cranial cavity, itself, to indicate many easily detectable extraneous "causes."

We have found in the pre-adolescent and adolescent group a tendency for the head, neck and extremities to grow rapidly, but the chest to be retarded (Fig. 2), so that the heart is small, the vessels of the neck and holes of entry into the skull infantile,

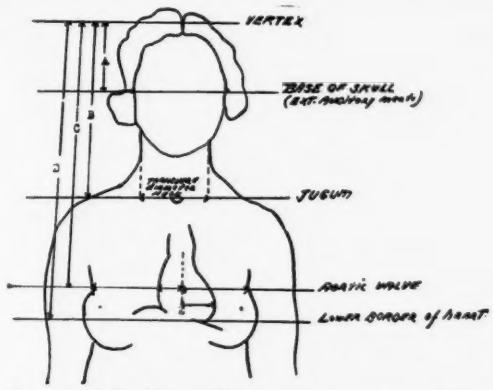


FIG. 3. Orthodiagraphic measurements as taken by flouroscopy. A, vertex to base of skull (external auditory meatus); B, vertex to jugular notch; C, vertex to aortic valve; D, vertex to lowermost border of heart; E, transverse diameter of heart.

and yet the demand for blood by the large growing brain organ is very great.

When a certain degree of disproportion is reached between the "pump" (heart, as a source of blood supply) and the brain, then trivial states of hydration, excitement, or overfatigue act as *precipitating* factors and may lead to a convulsive seizure which otherwise would not arise in a normally balanced individual.

Figures 4 and 5 illustrate the striking structural inadequacies which are to be found in a group of so-called idiopathic, institutionalized epileptics as compared with a similar age group of "normal" boys attending Y. M. C. A. gymnasium classes.

Alterations of blood flow to the brain, either by clamping both jugulars, simultaneously (Kussmaul-Tenner attacks) or by arresting the movement of circulation for from nine to seventeen seconds (Stokes-Adams syndrome) will frequently be sufficient to precipitate a convulsive seizure.

This fact is extremely important in the analysis of nocturnal attacks. Seizures at night are often unrecognized until the individual may find blood stained bedding or complain of a "sore" tongue or "sore" muscles, or some individual observe a true seizure.

Children frequently sleep on the abdomen and we have found several instances in which turning the head sharply to one side (in the presence of demonstrable anomalies of the venous outlets to the skull) compresses the "dominant" jugular vein, causing a rise in intracranial pressure and impairment of cerebral circulation as demonstrable by the "Tobey-Queckenstedt" test. In at least eight instances the convulsions have been entirely controlled by forcing the individual to sleep on the back or preventing acute flexion of the head and neck. This has been accomplished by devising a vest filled with a collection of corks, so that when the individual turns on to the abdomen, discomfort produced by the bag of corks prevents prolonged prone positions with jugular compression and at the same time the weight of this special

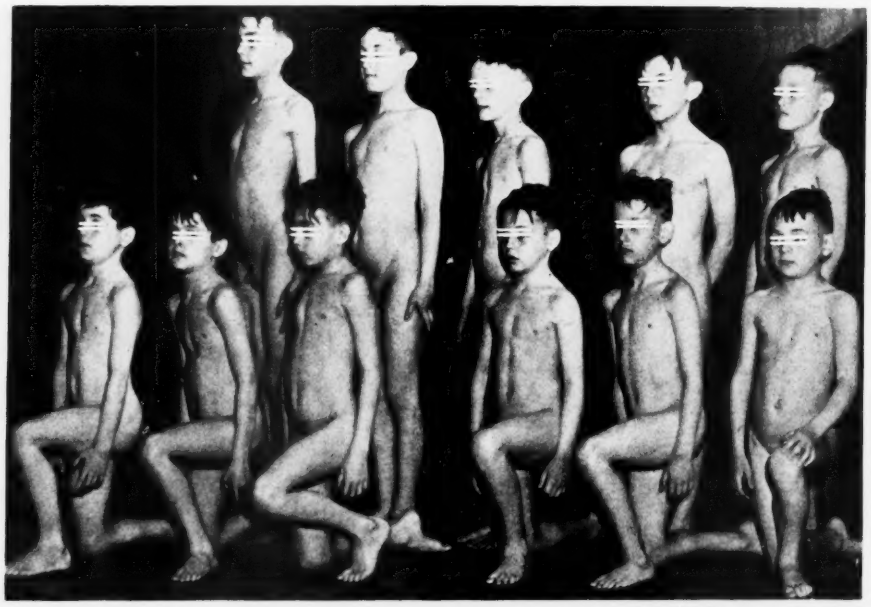


Fig. 4. Normal boys, six to ten years of age (Y. M. C. A. class). Compare chest, neck, head and extremity proportions with similar age group of structurally inadequate epileptics in Figure 5.

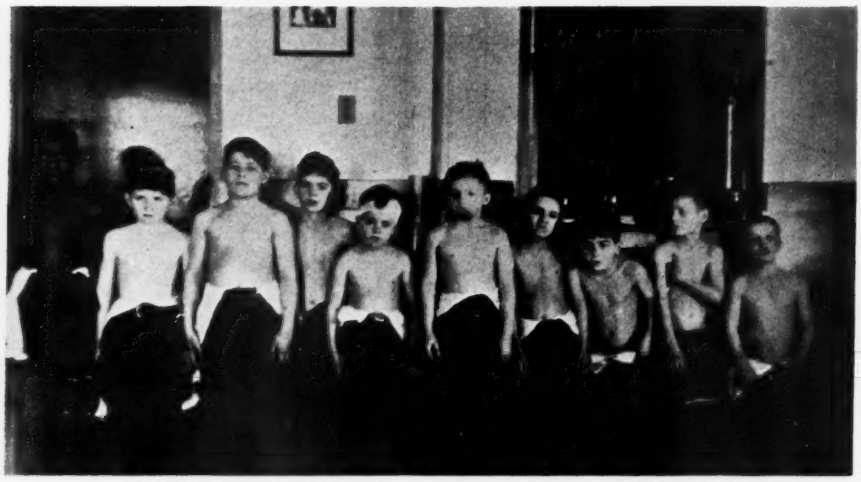


Fig. 5. Chronic, institutionalized, epileptic boys, six to ten years of age. Note marked disproportion between chest, neck, head and extremity development, high incidence of structural inadequacies and abnormalities. (Courtesy of Dr. A. W. Pigott, Skillman Colony.)

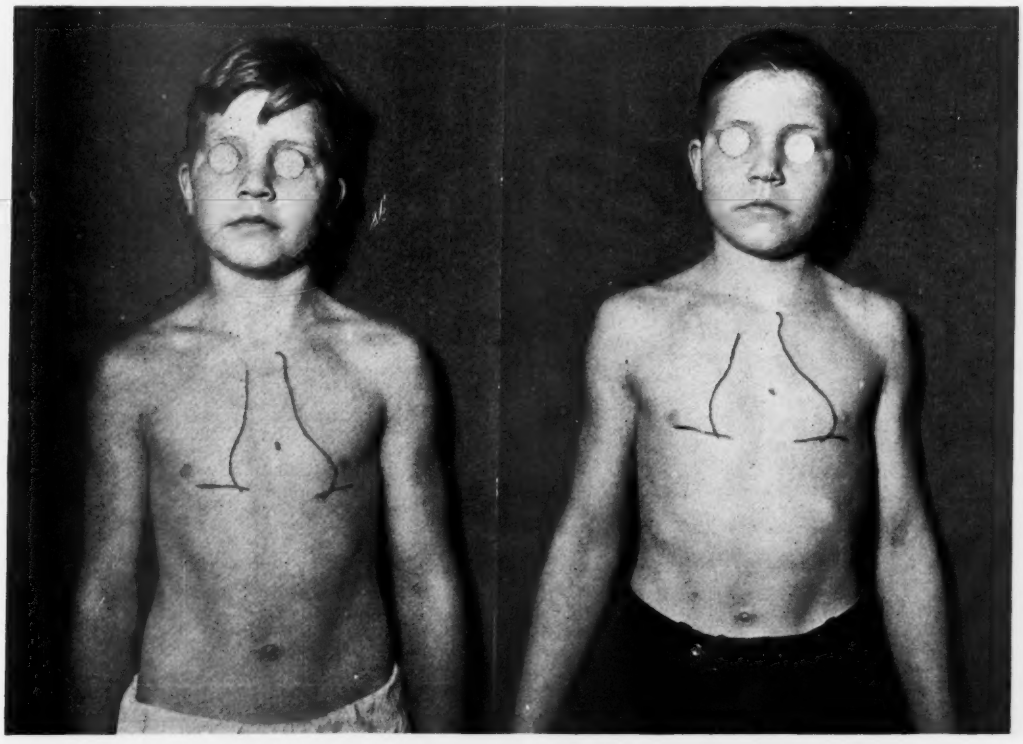
attachment is not sufficient to be disturbing, even to a child.

In considering the problem of "nocturnal" epilepsy, therefore, the clinician must observe or have detailed or illustrated to him the position in which the patient is inclined to sleep, where the hands are placed, and if during the relaxed state of sleep, postures may develop that would press on, kink, or obstruct venous return flow from the brain.

X-ray of the base of the skull (Chamberlain projection) for study of the vascular foramina; examination, measurement and determination of the size and development of the heart; estimating the distance between the heart and vertex as well as the size of the arterial structures will be necessary to evaluate the *degree* of *structural inadequacy*.

It will be noted that many of the adolescent types possess long, thin necks, small hearts and small vessels. Thus, the cardiovascular system is required to pump blood further, through smaller vessels into an otherwise normally appearing head and brain, and if occasional failure to sustain adequate circulation ensues, the period of arrested movements of circulation need only be similar to that noted under the "Stokes-Adams syndrome" to favor a seizure.

Correction, if possible, can be assisted only by appropriate exercises. We all recognize "athletic" heart as frequently arising from collegiate sports. Running, rowing, climbing, and appropriate daily activities, will do much to develop a larger cardiovascular system. (Fig. 6.)



A B

Fig. 6. G. M., male, age eleven. Grand and petit mal since five years of age, averaging as many as fifteen petit mal a day and one grand mal a month. Three grand mal attacks day of admission, August 31, 1938. Neurological examination and laboratory studies were negative. Physique suggests early structural inadequacy type. Encephalogram essentially normal pattern. A, discharged to out-patient dispensary on September 13, 1938 on special epileptic diet, fluid restriction to 16 ounces, phenobarbital gr. ½ twice daily and exercises to develop chest and heart. Since discharge there has been marked improvement, averaging only one petit mal and one grand mal every other month. Attends school regularly. B, note increase in size of heart and chest after six months of systematic exercises.

If the physician is willing to guide the patient through the period of adjustment from former sedentary life to one of forced activity, discounting temporarily the additional attacks of this period, he will find that such efforts will be rewarded not only by remarkable development of the chest and heart, but with proportionately gratifying clinical results from the standpoint of improvement in the mental state, the requirement of less drugs and with better control of the seizure.

SUMMARY

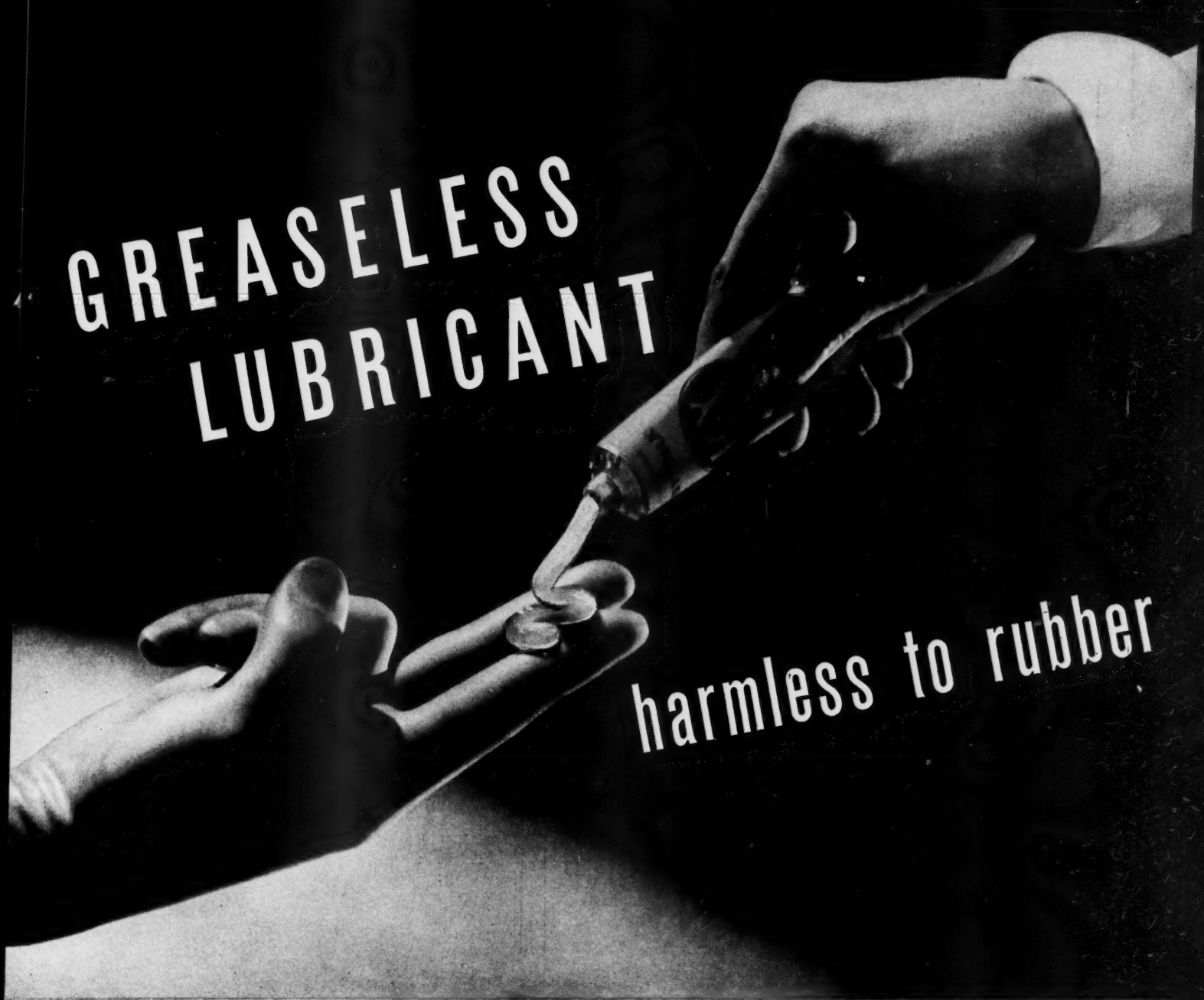
One objective in making the diagnosis is to recognize the attack or seizure as belonging in the field of the *convulsive state.* With this recognition, an attempt at classification of type must depend upon the concept as to the purpose and nature of the seizure.

If the concept of a "defense" reaction against a threatened failure of cerebral circulation is to be adopted, a search for those factors that directly or indirectly concern cerebral circulation, blood volume and pressure relationships should lead to a more accurate diagnosis as to the type, as well as to the direction indicated for treatment.

Where organic processes exist, surgery may be helpful. Where structural inadequacy exists, an active program of physical training, exercise, regulation of fluid, diet and appropriate medication will be beneficial.

Where the symptoms of convulsions arise secondary to some phase of "water intoxication," appropriate regard for the temporary fluid restrictions of the individual must be considered, as well as appropriate measures to enhance elimination through the skin, bowels, and kidneys, and thus prevent abnormally "high tides" of fluid being retained within the cranial cavity (reciprocal low tides of blood).

Finally, it may be pointed out that although fluids, *per se,* are not directly responsible for the seizure, they may displace blood volume and impair intracranial circulation to such a degree that the controlling centers are released more easily by appropriate stimuli and, therefore, certain individuals at certain times become more "vulnerable" to seizures where cellular permeability, altered brain metabolism and other organic and structural factors have also helped to *predispose* them to an attack.



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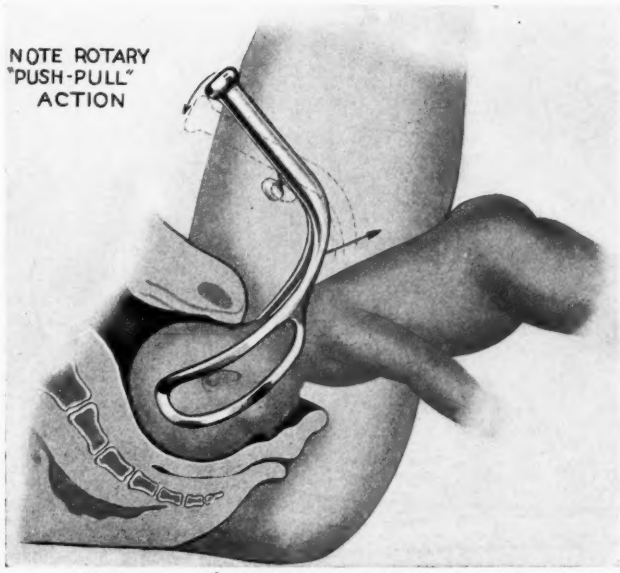

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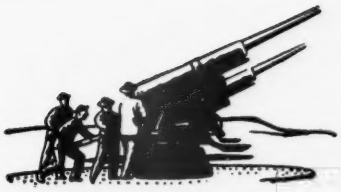

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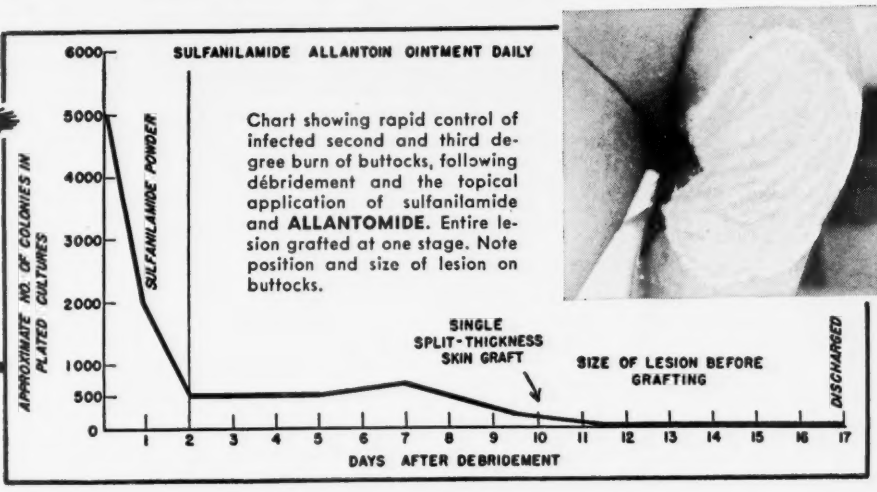

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*Veal, J. R.; and Klepser, R. G. (Surgery, Vol. 10, No. 6, Dec. 1941, 947)
**Veal, J. R.; Klepser, R. G.; and DeVito, Michael P. (Amercian Journal of Surgery, Vol. 54, No. 3, Dec. 1941, 716)

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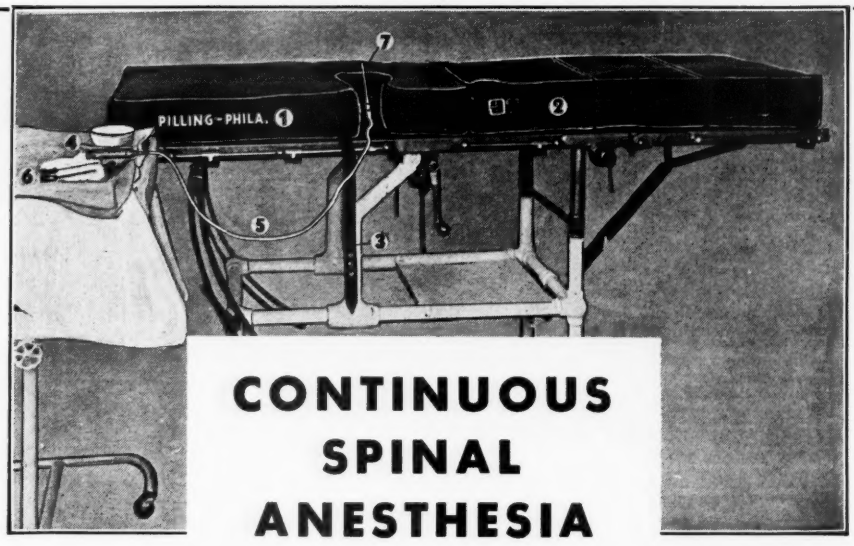

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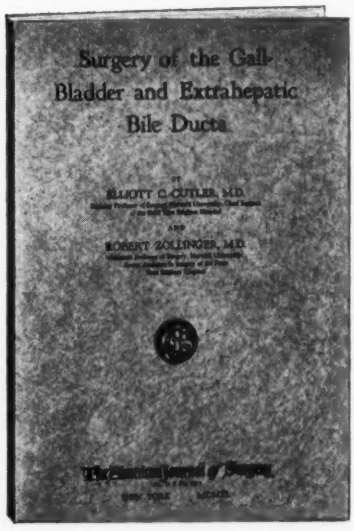

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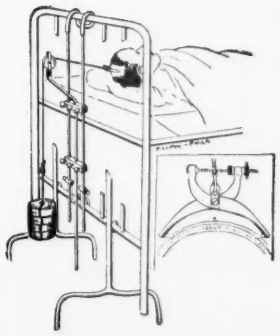

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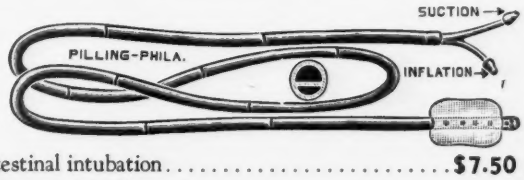

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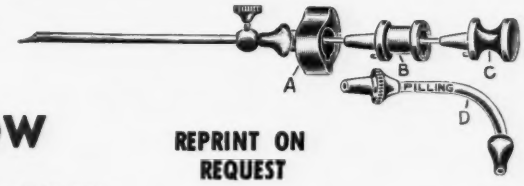

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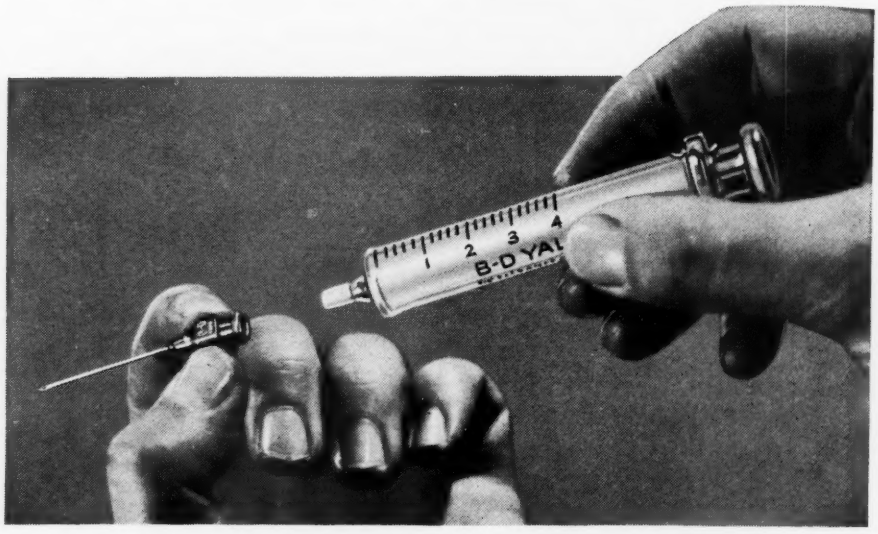

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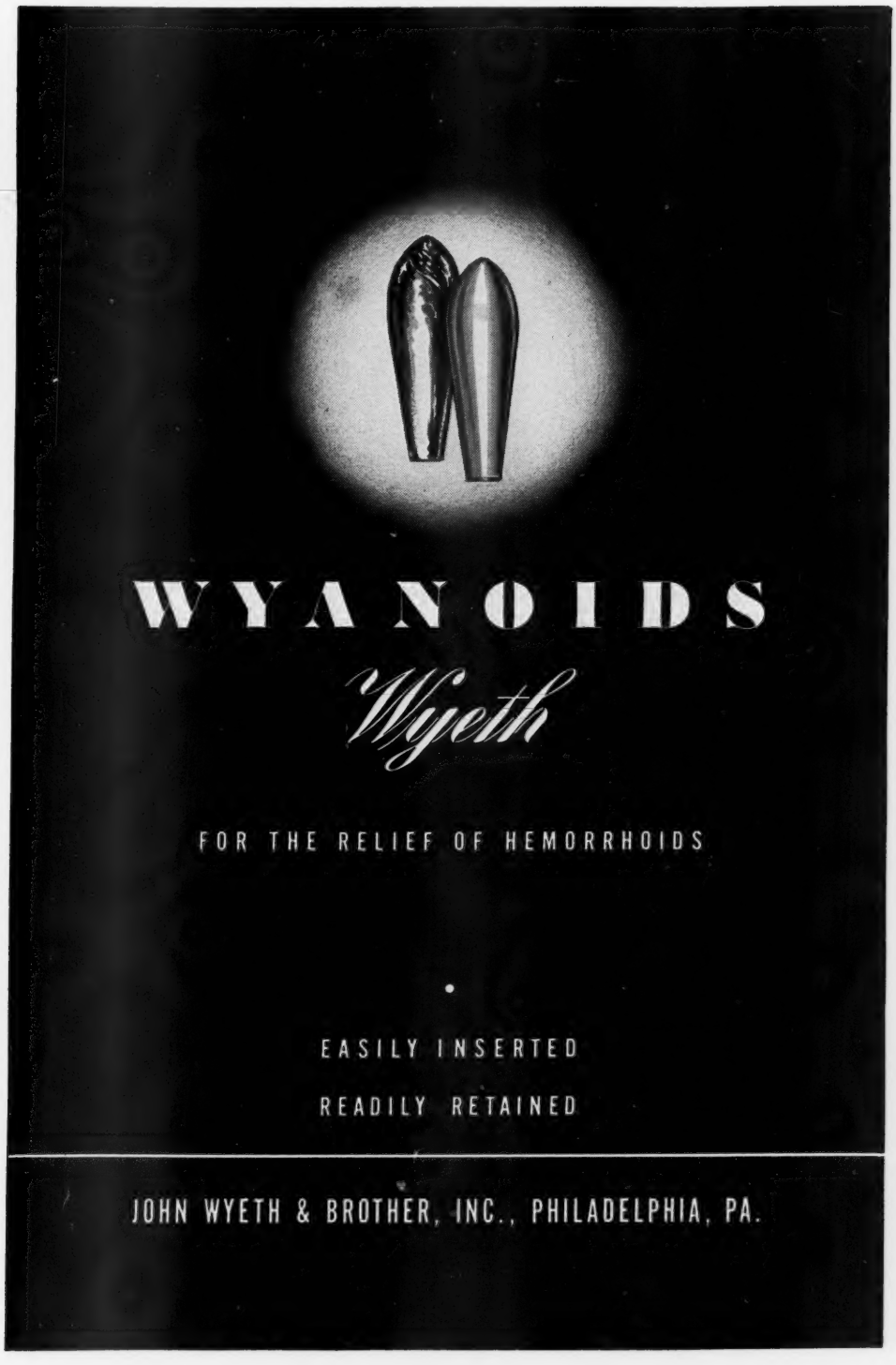
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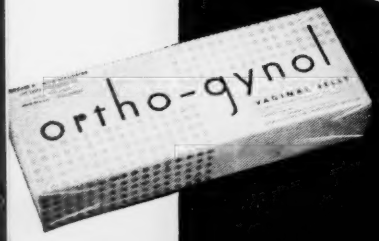

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